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BY

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PREFACE

Two years ago I was present at a meeting of a Medical Society where an obstetric physician read a paper on "Pregnancy Complicated by Heart Disease." To my surprise he made no reference to the great advances that have been made in cardiology during recent years; indeed, his attitude towards the subject was that of fifty years ago. It seemed to me, in fact, that the views expressed were not as recent as those of Angus Macdonald[1] when he published his treatise in 1878.

I turned to a number of books on obstetrics, thinking that possibly others had not been so negligent of progress; but I found the same condition of affairs.

I then felt that it was incumbent on me to do something to bring to many suffering women the benefits of modern medical knowledge. Some thirty years ago I had made a special study of the subject, but at that time I had found I could not get very far because of my imperfect understanding of the meaning and significance of the signs occurring in the various forms of heart affection. This imperfection of knowledge took

[1] "The Bearings of Chronic Diseases of the Heart upon Pregnancy, Parturition, and Childbirth."

me away from the immediate consideration of heart disease and pregnancy. Nevertheless the line of observation I pursued dealt with principles which could be applied to the pregnant state just as they could be applied to the circumstances of every-day life. As, however, no one seems to have applied these principles to pregnancy, I have now turned back to the notes and observations I made in the years during which I was engaged in midwifery practice. No one recognises better than I do how incomplete these notes are. I refer to them only because, so far, they seem to be the most helpful records available towards a recognition of the peculiar features of the problems under consideration.

I give them, therefore, with all their defects, and hope to show that, imperfect as they are, they can be of very real service when applied to this subject under the controlling influence of well-defined principles. But the main object I have is to direct the attention of obstetric physicians to the newer knowledge. I trust that this subject will be taken up and pursued by those who have greater facilities than I possessed, and that these workers will bring to bear upon the problem presented the great body of knowledge of heart affections which is now available.

J. M.

CONTENTS

CHAPTER IV

THE PHYSIOLOGY OF THE HEART IN RELATION TO ITS EFFICIENCY

CHAPTER V

THE LAW OF HEART FAILURE

CHAPTER VI

HEART FAILURE WHEN AT REST

CHAPTER VII

MURMURS

CHAPTER VIII

MITRAL REGURGITATION

CHAPTER IX

MITRAL STENOSIS

CHAPTER X

AORTIC DISEASE

CHAPTER XI

IRREGULAR ACTION OF THE HEART

CHAPTER XII

AURICULAR FIBRILLATION AND PAROXYSMAL TACHYCARDIA

LIST OF ILLUSTRATIONS

HEART DISEASE AND PREGNANCY

CHAPTER I

INTRODUCTION

THERE are few subjects in medicine of which an accurate knowledge is more urgently required than that of a woman's fitness for child-bearing. That a great strain is thrown upon the maternal heart during pregnancy and labour is well known. It is a matter of experience, too, that the pregnant state may throw more work on the heart than it can bear, with the result that the mother's life is lost, or her health irreparably impaired. The child, too, often dies. These disastrous happenings surround a natural process with dread and mystery, for the dread is aggravated by the fact that the source of danger is not clearly realised. As a result all sorts of signs are looked upon with suspicion—signs innocent as well as signs grave. Needless to say, this obscurity does a great deal of harm. Many women are subjected to unnecessary alarms and restrictions when pregnant; others have to suppress the natural desire of motherhood; while connubial relations are often disturbed by

warnings and hints of dangers which may never arise.

Defect in Medical Knowledge

This unsatisfactory state of affairs is entirely dependent on a defect in medical knowledge which it is quite possible to remedy. That the matter is not worse is in a measure due to the fact that the vast majority of women, especially women of the working-classes, never have their hearts examined. Were they systematically examined, abnormal signs would be discovered in many instances; the significance of these signs would not be understood; grave doubts would be aroused. Thus, many who have passed unscathed through the pregnant state would have been unnecessarily frightened and alarmed.

When we seek for an explanation of our ignorance on this subject we find that it is due to the fact that medical knowledge has not advanced far enough to enable us to understand the meaning and significance of certain physical signs. Moreover, we find a new difficulty when we come to ask ourselves in what way our lack of knowledge can be made good. Gradually it is forced on our minds that if we would understand clearly defects and their origins we must go back to the beginning and consider the way in which, at present, the subject-matter of these chapters is investigated and taught.

The teaching of disease of the heart comes

within the province of the physician. When, however, one comes to estimate the opportunities the physician has for dealing with the relation of heart disease to pregnancy, one soon discovers that his practice never brings him into contact with pregnant women except when one of these latter is referred to him for an opinion. On these occasions it frequently happens that instead of frankly recognising that his experience does not justify him in claiming a knowledge of the subject, he gives a prognosis of the "guarded" type. He says in effect: "The patient may undergo pregnancy safely; on the other hand, there is a possibility that she may not." The effect of this is that the patient is left with a vague notion that some dread fate hangs over her head. In other instances the physician "plays for safety," and forbids the pregnancy, yet these patients may be perfectly capable, in many cases, of undertaking it.

The obstetric physician, if we may judge from modern textbooks, has never taken the trouble to understand the elements of cardiology; in consequence he continues to pursue his enigmatical methods. While the advances in cardiology are barely mentioned in books on general medicine, they are not dealt with in books on obstetrics. In the recent textbooks on this latter subject which I have read there is not the slightest indication that any advance has been made in the study of the heart during the last forty or fifty years—the writers apparently being unconscious of the revolutionary changes which

have taken place in our knowledge of this organ in this period of time.

I am emphasising this point for reasons which have only an indirect bearing on the matter in hand. I wish my reader to visualise clearly the methods peculiar to medical investigation. Pregnancy is not the only condition which is shrouded in darkness. There are a great many other questions of a similar kind which are an integral part of the practice of medicine and which await inquiry. No progress, I believe, can be made in regard to them till it is understood how medical investigations should be conducted. The matter of heart disease and pregnancy serves thus as an illustration of a wider difficulty. It shows, if we will but learn the lesson, how completely our present-day method of medical research fails of its purpose. It is my aim to indicate the nature of this failure, and to explain in what manner I conceive that success may be attained.

Personal Experiences

My attention was early directed to the subject of Heart Disease and Pregnancy by a tragic experience which occurred soon after my entrance into general practice, about forty years ago. I attended a young woman, thirty-five years of age, for a miscarriage at the third month of her first pregnancy. I discovered then that she had a presystolic murmur ; so far as my investigation of the condition went, there was nothing more the matter with the heart. I had a vague notion

that mitral stenosis was a serious embarrassment to the heart in pregnancy, but of the source of danger and its nature I had no knowledge. The woman left my neighbourhood for a year, at the end of which time she returned to her father's house, seven months pregnant, and with a considerable œdema of the legs. This œdema increased until, when labour set in at full time, she was much swollen not only in the legs, but in the abdomen. The external genitals were so œdematous that an examination per vaginam could be carried out only with difficulty. As the labour proceeded little progress was made; the suffering was very great, and there was marked distress in breathing. The patient had to lie propped up in bed, a position which rendered the use of instruments impossible. Moreover, so profound was the sense of suffocation that attempts to give chloroform were resisted. Two practitioners of much experience saw her, but after many hours of suffering she died undelivered, from extreme exhaustion.

I had, at that time, a fairly large midwifery practice, and, on several occasions during the next few years, I had some rather trying experiences in attendance on women with heart trouble, though no other case proceeded to such a disastrous ending. I noticed, however, that in a number of patients with damaged hearts the pregnancy further impaired the functional efficiency of that organ. The importance of this being thus forced upon me, I read carefully all the available literature. This, indeed, confirmed

the view I had formed as to the danger in mitral stenosis, but gave me no enlightenment about the risks in other forms of heart affection. There were many vague hints of the evils arising from valve lesions, but of sufficiently definite information to enable me to recognise which types of these lesions were to be feared there was none. Even the changes occurring in the normal heart during pregnancy were very imperfectly described.

State of Knowledge of Cardiac Phenomena

I made a note of the results of my reading, and propose to give the gist of it here as a kind of review of the state of knowledge of cardiac abnormalities prevalent forty years ago. That this may be found strangely apposite to-day by certain of my readers will only show, I fear, that the attitude of the profession of those early times is still the attitude of the bulk of teachers and authors. Indeed it seems to me, as I turn back to these old pages, that the trend of modern medicine, both in teaching and investigation, is to render the solution of such problems as the relation of pregnancy and heart disease impossible. Before the discovery of auscultation and the use of the stethoscope, about 100 years ago, experienced physicians estimated the state of the heart by its manifest functional efficiency ; and in reading the authors before that date, I find that many of the views which I have arrived at were anticipated by them. There is no doubt

in my mind that the line pursued by those physicians was sounder than that which has been followed since the introduction of mechanical devices. The coming of the stethoscope was the beginning of a method in clinical research which has greatly hampered the practice of medicine, in that the introduction of mechanical methods has led to a confusion as to the kind of knowledge which these methods are capable of affording. This criticism applies not only to the stethoscope and its various modifications, but to all the other instruments which have since been employed in the examination of the patient. I need mention only the sphygmograph, polygraph, electro-cardiograph, and blood-pressure instruments.

The discovery of the sounds of the heart and the various modifications of these sounds was followed by a period in which a great deal of time was spent on auscultation and on the discussion of the phenomena revealed by it. At first the cause of the cardiac sounds was not clear ; a long series of observations, experimental and clinical, was therefore undertaken, to find out the manner in which the sounds were produced. I do not dispute that a great deal of valuable knowledge was thus accumulated.

One unfortunate consequence of this concentration upon the new method, however, was to give it a preponderating importance in the examination of the heart, and to minimise the value of the study of other phenomena, with the result that unjustified conclusions were drawn from mere modifications of the sounds. This, as

has been indicated, was an error inherent in the view taken, at that time, of the possibilities of the new instrument. The physician who detected an abnormal sign supposed that in detecting it he had obtained information of a sort hitherto only obtainable by a test of functional capacity. He believed that he had gained knowledge entitling him to make a prognosis without the labour of determining how far the patient's activities were already curtailed. What he failed to see was that so long as he did not know the mechanism of production of the abnormal sound, nor the after-histories of people who might have this abnormality, he could not properly base conclusions on its presence. Thus, in reading the early literature of heart affections, since the introduction of the stethoscope, we find a grave significance attached to the presence of a bruit, at a time when the cause of the bruit was unknown and the kind of information which it conveyed was not understood. I have sought for an explanation of this rather astonishing state of affairs. I find it, I think, in the probability that a physician, examining dying patients in whom a bruit was present, would attribute the failure of the heart to the cause of the bruit. That this is the correct explanation is rendered more likely because we find similar reasoning following the discovery of a sign by any new method. Thus, to-day, physicians and "heart specialists" may be observed attributing serious significance to peculiarities detected in graphic or electro-cardiographic records or in blood-

pressure readings before they understand the nature of the signs on which their gloomy forebodings are based.

I dwell on this to draw attention to the fact that medical knowledge has not even yet advanced so far as to enable investigators to understand the principles which should guide them in recognising the significance of an abnormal sign. They do not know how to apply their observations in practice, nor what steps to take to obtain an estimate of their significance. This statement may seem to be an exaggeration; if, however, the reader will consider the methods, to be described later, which I was compelled to employ in order to find out the significance of a symptom, he will, I think, realise not only that a defect in knowledge existed, but also how it came to exist; and why it is still with us.

The hasty conclusions formed by the early observers in regard to the significance of murmurs has hung like a mill-stone round the necks of the profession; to-day these conclusions mislead the vast majority of doctors, and cause much unnecessary trouble and injury to people in many walks of life.

CHAPTER II

THE NORMAL CHANGES IN THE MATERNAL CIRCULATION DURING PREGNANCY, CONFINEMENT, AND THE PUERPERIUM

Methods of Investigation

In my endeavour to find out wherein lies the danger of pregnancy to women with heart disease, I experienced most difficulty in determining how to set about such an inquiry. The study of patients with damaged hearts proved fruitless and misleading, for I was not able to tell the significance of some of the symptoms. I had already supposed certain of these latter to be abnormal and of grave significance, on grounds which I was coming to recognise were insufficient. At last I realised that it was necessary for me to understand the phenomena which occur in healthy women if I was to form a clear idea of the changes taking place in consequence of the accommodation of the circulatory system to increased work.

I began, therefore, to observe women during their confinements, noting all the signs which I took to be departures from the normal, and also those which occurred as a consequence of the straining during the labour. I soon found quite a number of changes; for example, modification

of the sounds, murmurs, irregularities, displacement of the apex beat outwards to the left, some duskiness of the countenance, and so on. Some of these signs were modified or disappeared during the puerperium. I was at a loss to explain their presence or their significance, and I saw that the subject required far more thorough investigation. I recognised that it would be necessary to watch the onset of these signs, and the conditions or factors which caused them; then, it would be necessary to watch patients for a long time after their confinements to see if the signs disappeared or remained to give rise to trouble in the future. This latter part of the problem was one of extreme difficulty. For it was not only necessary to note the progress of the patient, but also to find an intelligent method of estimating the significance of the changes which might be observed.

In the routine of my work as a general practitioner, I had opportunities of seeing women before they became pregnant. I made careful notes of the state of the heart and circulation in such of these as I expected would become pregnant. When they consulted me afterwards for early troubles, such as sickness and vomiting, I examined their hearts at intervals. Further, I examined those who bespoke my service for their confinements, and kept them under observation till labour began. As time went on, I found that one sign after another required particular observation, and so I had to adopt special methods to obtain the information I desired.

Thus I found œdema of the lungs to be a very important matter in heart failure, and its relation to the pregnant state had to be specially considered. I found, too, that the embarrassment of the circulation increased during the last months of pregnancy, and that a number of new symptoms appeared then; for example, murmurs, irregularities, and pulsation of the veins of the neck. It was necessary to know exactly when such changes began; so I used to ask the patients to stay in bed on certain days till I had visited them. The different points were carefully noted. Finally, the significance of these signs, as they were related to the heart's efficiency, had to be considered. This led to an inquiry, extending over many years, which is not yet complete.

I will deal specifically with the more important symptoms in later chapters; here I propose to summarise the results of the alterations which occur in healthy people.

The Changes during the Early Months of Pregnancy

Practically no changes in the circulation could be detected in the early months of pregnancy. Sometimes sickness confused the picture, bringing in its train exhaustion, and variations of the pulse rate, but as time went on these disappeared.

Limitation of Response to Effort

Towards the sixth month the response to effort began to be restricted, i.e. breathlessness

appeared after an amount of exertion which had hitherto been accomplished in comfort. As a rule this never became extreme, and the woman was able to attend to her household duties; she felt the breathlessness chiefly on going upstairs.

Effect of Increase in Size of Uterus

About the seventh month the uterine tumour altered the shape of the chest—widening it out so that the circumference gradually increased. The chest was measured immediately under the breasts and, as full time approached, the circumference increased considerably in some cases. Immediately after delivery the circumference diminished, and day by day it decreased till some weeks after confinement it had returned to normal.

The following are a few examples:

Case 1

	Inches.
At full time	28
Immediately after birth of child .	25½
Four days after birth of child .	24½

Case 2

At full time	26½
Immediately after birth of child .	26
Seven days after birth of child .	26

Case 3

At full time	29
Immediately after birth of child .	26½
One day after birth of child . .	25½
Three weeks after birth of child .	23½

Displacement of the Heart

Coincident with the change in the chest circumference the heart was frequently displaced until the apex beat was pushed out one inch beyond the left nipple line, and upwards to the fourth interspace. After labour the heart gradually swung back into its normal position.

Hypertrophy of the Left Ventricle

It is well to bear this normal displacement in mind. For a long time it has been held that the left ventricle hypertrophies during pregnancy and undergoes some sort of involution after confinement. The evidence for this view has been based chiefly on theoretic grounds, on some very imperfect post-mortem observations, and on the displacement of the apex. Further, the safety of chloroform anæsthesia during pregnancy has been attributed to the same circumstance. No evidence of hypertrophy could be perceived (see Chapter IV, p. 30).

Stasis of the Lung

Accompanying this widening of the chest, there is a tendency for stasis to occur at the basis of the lungs. This will be dealt with in speaking of the failure of the right heart. In order to estimate the factors which embarrass the right heart, the lessened effect of the movements of respiration had to be considered. I found that normally there was a tendency to stasis even in

healthy women when pregnant. In the periodic examination of pregnant women I found that a few of these, after lying in bed all night, had crepitations at one or both bases. These crepitations disappeared after one or two deep breaths, and were never present during labour. They returned for a few days after the confinement. The meaning and significance of this sign will be discussed later (p. 49).

Venous Stasis

A variety of phenomena appeared on the venous side of the circulation. In a number of patients the veins of the legs became swollen and varicose—in some there was considerable swelling and varicosity of the veins of the thigh, and of the vulva. Hæmorrhoids were frequent. The cause of these changes is not clear to me. I lean to the view that they are the result of actual pressure on the veins in the pelvic or abdominal cavities. Some writers, however, say that these signs may appear at a stage so early that pressure is out of the question. I had not myself observed them at such an early stage. There is, no doubt, a marked change in the peripheral vascular system in the smaller arteries and veins, which is made evident particularly in the breasts. The mammary artery, usually imperceptible, becomes dilated, and probably the arteries generally dilate to a slight extent, though this is not capable of measurement. One can infer so much from the fact that many women

become fatter during pregnancy. There is perhaps also an increase in the quantity of blood.

I find that some writers attribute the hæmorrhoids and varicosities of the vulva to back-pressure from the heart. That is an error, of little importance so far as this matter is concerned, but one which must be pointed out, as it misleads when we are dealing with heart failure. The idea that the heart fails by an engorgement affecting one chamber after another has long held the field. We are invited to picture regurgitation from one orifice after another until the right auricle and ventricle throw blood back into the venous system, causing the appearance of venous stasis, varicosities, and dropsy. While no doubt there is a little truth in the idea, it can be laid down that such an effect of heart failure has only a very slight influence and is not the cause of the phenomena attributed to it. I have seen many people die slowly of heart failure in whom none of these signs appeared. I have seen the right auricle and ventricle throw such quantities of blood back that the liver was distended and pulsated at each systole; yet there was neither venous stasis nor dropsy in the legs. Experience shows that varicose veins are frequently present in people with perfectly healthy and efficient hearts.

Pulsation in the Jugular Veins

Another symptom usually, but erroneously, considered to be the result of this back pressure is pulsation in the veins of the neck. During a

systematic inquiry into the signs exhibited by the circulatory system my attention was arrested by the movements in the veins of the neck. At the time when I began the inquiry very little attention had been devoted to this subject. The

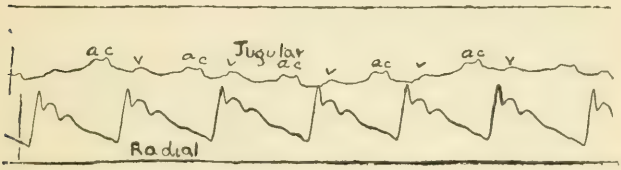

FIG. 1.—Tracings of the jugular and radial pulses the day after the confinement, showing only a faint movement in the jugular tracing.

pulsations indeed had been recognised, and graphic records of them had been obtained, but the nature of the movements taking place and their significance were not understood.

Among healthy women during pregnancy I

FIG. 2.—Tracings of the jugular and radial pulses the second day after confinement, showing a large movement in the jugular tracing.

found that these pulsations vary in an extraordinary way. They are present at one time, and absent at other times; sometimes they are small and barely perceptible (Fig. 1), then again they become of great size (Fig. 2). The records of many patients showed that pulsations were

often quite distinct before pregnancy. They might remain unaltered during the whole period till labour set in. During labour they practically always disappeared, but returned in a day or two after delivery, sometimes attaining a great size. Thus in the case from whom Figs. 1 and 2 were taken there was no jugular pulse during the labour, a faint pulse next day (Fig. 1), and a large pulse on the second day (Fig. 2). I attempted to correlate these variations with coincident changes in the heart—seeking for evidences in the size of the organ, in its sounds, or in its functional efficiency, but I failed to find any relation. I thought that the increased size of the venous pulse might indicate a dilatation of the right heart, but I found the increase taking place and the heart remaining normal in size, with no alteration of the cardiac sounds.

The disappearance of the venous pulse during labour seemed capable of a reasonable explanation. A false view of the effect of effort upon the heart has long been held—the view that the healthy heart dilates with effort. More careful observation reveals the fact that, instead of dilating, the healthy heart actually becomes smaller on effort. It will be realised, if thought is given to the subject, that this is to be expected. When effort is made dilatation takes place in the peripheral vessels. The result is that during effort there is, in the peripheral vessels, a relatively greater amount of blood than is present in them when the body is at rest. As the size of the heart depends partly on the quantity of its

blood-content, with a diminution of the blood during effort the size naturally becomes smaller.

The right auricle and the veins leading to it act as a reservoir to the ventricle. The pressure in the veins and auricle is never of itself able to distend the ventricle and cause dilatation; so that, when there is a free return of blood from the periphery, the jugular vein is never quite empty. The emptying and filling of the auricle and ventricle cause a variation in the quantity of blood in the jugular vein, and it is this which we recognise as the pulsations referred to, and which we can graphically record (Fig. 2). When, therefore, a larger quantity of blood is contained in the peripheral vessels, the jugular vein does not receive a sufficient amount for the movements occurring in it to be perceived. After the effort has ceased, however, the returning blood increases to such an extent that the reservoir becomes filled. The movements are then again visible.

In order to comprehend the relation of these pulsations to heart failure I watched patients with a jugular pulse for many years, noting the effects of effort and illness—particularly febrile illness. In rare cases I discovered great pulsation in the veins with certain forms of heart failure—but, on the whole, I found that neither the presence nor the absence of pulsation was of any use in estimating the efficiency of the heart. In those rare cases to which I have referred there were present other signs which gave a surer indication.

Where, on the other hand, the study of the jugular pulse became of the greatest value was in unravelling the mechanism of cardiac irregularities. By observing the changes in the jugular pulse, I discovered a method of differentiating between the waves produced and of relating them to their causes in the heart itself. This matter will be referred to in dealing with Heart Irregularities (Chapter XI, p. 92).

Dropsy

The mechanism by which dropsy is produced is still one of the most obscure matters in medicine. My notes of cases of pregnancy show that in healthy women, at this time, swelling of the legs was far from infrequent. It was usually slight, but sometimes became very considerable. As it tended to get worse towards the end of the pregnancy, and as there were no accompanying changes in the heart, I attributed it to the pressure of the uterus on the veins. At all events, the women went through their confinements quite well, lived for years afterwards, and bore other children. Even those women who also had albumin in the urine did well. (I am not referring to cases of albuminuria which ended in uræmia. I had, like every other general practitioner, experience of these. My opportunities, however, were too restricted to enable me to get a full knowledge of the significance of this sign, which, be it said, still awaits elucidation.)

Cyanosis

During labour, in many patients, the face becomes tinged and dusky, while the lips become of a dark red. I made a great many observations on the capillary circulation in this connection, but nothing definite was elicited.

In many women systolic murmurs and irregularities appeared and disappeared. These will be dealt with in subsequent chapters.

CHAPTER III

CHANGES IN THE DISEASED HEART IN PREGNANCY

Abnormal Signs appearing during Pregnancy

I had many patients who showed abnormal circulatory signs. Let me say at once that the great majority of these passed through the pregnant state, confinement, and puerperium with no trouble. Thus, some cases of mitral stenosis did not seem to suffer in the least, while others suffered severely. Cases of irregularity showed the same variation—the great majority passing safely through, a few revealing signs of heart failure. Cases, again, in which the heart was weakened from other causes, but where there were no abnormal physical signs such as murmurs or irregularity, bore the pregnancy well and seemed little the worse afterwards. These latter were usually breathless and the victims of precordial pain.

The Interpretation of Abnormal Signs

At the time when I was making these observations, the results seemed very bewildering; they did not enable me to recognise where the

danger lay—which was the object of my research —and their variations seemed without principle. I had forced upon me the urgent necessity of a more thorough knowledge of the fundamental nature of abnormal signs, the mechanism of their production, and the bearing of their causes on the heart's efficiency, especially under stress. To obtain this knowledge I began a new, long investigation into the meaning of all manner of cardiac symptoms. This investigation has occupied me for many years; it took me away from the study of heart disease and pregnancy.

I missed, therefore, the opportunity of studying directly heart failure in pregnancy in large numbers of women. On the other hand, I obtained some knowledge of the fundamental principles underlying the production of heart failure. The application of these principles to the cases of heart disease complicating pregnancy which I have since encountered has, I believe, demonstrated their usefulness. It has demonstrated also the value of the type of observation of which I am speaking. I had, as a consultant in London, a great many women referred to me by doctors who were anxious to know whether they were fit for child-bearing or whether, pregnancy having been incurred, there was danger. In these instances I made use of the principles I am about to describe. They guided me to an opinion. The results fully justified the faith I had learned to repose in them.

The Differentiation of Abnormal Signs

In order to acquire a knowledge of the source of danger in heart disease complicated by pregnancy, I had to get a clear view of the heart's work, how this organ accomplished its work, and what were the signs when it failed to perform its duties in an efficient manner. I conceived that the key to this problem was heart failure. I searched the literature of the subject, but except when heart failure had reached an extreme degree, giving rise to dropsy, dyspnœa, and enlarged liver (sometimes spoken of as the "cardinal signs of heart failure"), little attention seemed to have been devoted to it. Indeed, so confused were the descriptions that no certain differentiation appeared to have been attempted between the phenomena of heart failure and those of other, accompanying but merely incidental, states. Nor was there any clearer distinction to be found between signs which indicated disease and signs in their nature physiological, or so little pathological as to be of no prognostic significance. Thus, it was recognised that murmurs or irregularities might in many cases be indicative of nothing serious. It was stated that there were other murmurs and irregularities which were of grave significance. What was not stated was how one might differentiate the serious from the trivial, the important from the merely incidental.

In the case of irregularities matters were in worse plight. Here there was no differentiation at all. I had not gone far into this subject before

I found that progress was impossible unless and until I should be able to distinguish one form of irregularity from another.

The Significance of Abnormal Signs

Even when, at last, I had succeeded in recognising clearly the different forms of irregularity there remained the important question: "What bearing does their presence have on the patient's future?" This question applied, as I saw, with equal force to the subject of murmurs. Attempts to answer it had in this latter instance been made, but they had not succeeded, for the matter was left in a very vague and indefinite state.

The question is one of such urgent moment to the rational practice of medicine that only the slightest consideration is required to convince anyone that a solution of it must be obtained. Nevertheless, I repeat, no really systematic endeavour to find a solution had been made. The prognostic significance of such common signs as murmurs and irregularities was by no means understood. The somewhat rule-of-thumb procedure which led to the vague notions then prevalent hindered as often as it helped.

The Misunderstanding of Prognosis

When I reflect now upon the steps I took to obtain this knowledge I can understand to some extent why it had been neglected. Prognosis, at first sight, seems an easy business as compared with diagnosis. Moreover doctors, as students, have their prognosis given to them ready-made

in terms of diagnosis. They are told, for example, that a murmur means a damaged heart. As soon as they are out of school they are placed in positions which impose the necessity of making prognoses. If they enter the army or navy they are at once put to the duty of examining recruits, a matter requiring a knowledge of prognosis. If they become examiners for life insurance the same knowledge is essential to the performance of their duties. In every field of medicine indeed where an opinion as to the future of an individual is concerned, the assessing the prognostic value of signs and symptoms is necessary. Thus the young doctor begins at once to act upon what he was taught. He accepts the ready-made prognosis and gets into the habit of using it. He says that "a murmur means a damaged heart," and does not doubt that he is right. Thus prognosis seems to be a matter of the greatest simplicity once a diagnosis of signs has been arrived at. The vast and vital difference between the sign itself and the nature and significance of it is overlooked or forgotten.

It is scarcely necessary to dwell upon the importance of a true knowledge of prognosis to a doctor who has to determine whether a woman is or is not fit for child-bearing.

Method of Arriving at Prognosis

In my own inquiry into prognosis in heart affections I made many false starts before I recognised the principles which should guide me. One day, however, and quite suddenly, I put to

myself the question "What are you afraid of?" The reply was, of course, "Heart failure." At once there came the next question: "Do these signs indicate heart failure, or do they foreshadow its occurrence?" This brought home to me my lack of knowledge.

I realised at least, clearly, the difference between the mere recognition of a sign—diagnosis—and its interpretation. I saw that prognosis hitherto had been accomplished by a process akin to jumping over an obstacle. One found a sign and called it a disease, instead of attempting to elucidate the mechanism of its appearance, or labouring to watch the responses to the calls of life of the man or woman showing it. That kind of "prognosis" was a double guess. One guessed that a murmur meant heart disease, and then one guessed again that heart disease meant an early death. Thus there was forced on me the need to watch and to follow individuals with all forms of cardiac abnormality year in and year out. To study them in health and in periods of acute illness. To be with them during prolonged bodily exertion. To note, if possible, the very earliest signs of heart failure, and to observe every variation of these signs during the years of their development. To make myself familiar with periods of improvement and with periods of regression. Finally, to be present if patients died and to undertake post-mortem examinations. These labours I made some attempt to carry out. Their results will be detailed in the following chapters.

CHAPTER IV

THE PHYSIOLOGY OF THE HEART IN RELATION TO ITS EFFICIENCY

The Adaptability of the Heart to the Body's Needs

The function of the heart is to supply blood to the body. The amount of work done by the heart is not constant, for it has to vary its activities according to the requirements of the different organs. The organs of the body vary in their activities. At one time they are functioning; at other times resting. The heart also varies its activities to meet these requirements.

The work of the heart is greatly facilitated by the modification of the function of other parts of the circulatory apparatus. Thus, during the activity of an organ the heart's output is increased, chiefly by an increase in the rate of its contraction; at the same time its work is facilitated by relaxation of the blood-vessels of the active organ. The heart itself receives a greater supply of blood during its increased activity. Starling computes that the amount of blood received by the heart during effort may be three times as great as that which it receives when at

rest. This increased flow is rendered possible by the peculiar distensibility of the coronary artery.

The Rest and Reserve Forces of the Heart

The ability of the heart to accommodate its work to the requirements of the body is due to certain qualities inherent in the heart muscle. When the body is at rest the heart supplies just that quantity of blood which maintains existence. When effort is made it has the power of increasing its energy to meet the new demands. There are thus two forms of Heart Force: one sufficient for the needs of the body when at rest —the Rest Force; and another, held in reserve, and used only when an effort is made—the Reserve Force.

While this distinction is somewhat theoretical, and is not sharply defined, yet the difference between the heart at rest and the heart during exertion is a very real one. Its importance will be made clear when we are considering heart failure.

The Response to Effort in Health

The healthiest heart can be forced to do only a certain amount of work; in other words, the reserve force is limited. Exhaustion of the reserve force is shown by signs of distress, particularly by dyspnœa. When effort is made the heart responds and continues to use its reserve force; so long as this is not exhausted effort causes no distress. Thus though an individual

may be able to walk a certain distance in comfort, if he carries a heavy weight he will suffer distress before that distance is accomplished. His heart is not in any way impaired, but its reserve force is exhausted sooner.

The Relation of the Reserve Force to Pregnancy

This simple statement explains many of the phenomena which occur in the heart during pregnancy. We know that this state imposes more work on the heart, and this not only in connection with the maintenance of the placental circulation, but also in respect of the additional weight carried by the mother. There are again disturbing factors in the form of interference with the shape and movements of the chest and displacement of the heart itself.

All this additional work is met by a call upon the reserve force. This is shown by the fact that the field of response to effort becomes limited; the pregnant woman cannot run upstairs so freely or so easily as was her wont.

Does the Left Ventricle Hypertrophy during Pregnancy?

The assumption that the heart hypertrophies during pregnancy is, as I have suggested already, not justified. To begin with, there is no need for hypertrophy so long as the reserve force is ample; as we shall see, when it is not ample, grave complications arise. Moreover, it is a

mistake to imagine that a heart can hypertrophy in the easy fashion postulated. While it is possible that long years of continued severe effort may produce a slight increase in the size of the organ, this increase is so small as not to be perceptible in the vast majority of people who lead strenuous lives. It is a matter of observation that strong, healthy, vigorous men do not have larger hearts than men carrying on much less vigorous work. The fact that pregnant women are hampered by breathlessness in making some effort which previously they could undertake in comfort shows an earlier exhaustion of the reserve force. This does not suggest hypertrophy. Further, hypertrophy cannot be detected by a physical examination of the heart, except in those instances—for example, aortic regurgitation—in which it is very well marked. In such cases there is a big and forcible apex beat.

This forcible apex beat does not present itself in healthy pregnant women. The changes in the shape of the heart which occur are thus not due to hypertrophy. On the contrary we have a complete explanation of these changes in the displacement of the organ and the widening of the chest caused by the uterine tumour.

The Relation of a Normal to an Impaired Efficiency

The distress arising from an exhausting effort may not be due to disease of the heart, for distress can arise in health. This is well seen when

any one becomes "winded" on running as far as he can. So that the phenomena of exhaustion are the same in quality in health and in disease. In health they appear more slowly, in disease more rapidly.

The distance which a healthy person can run is determined by the onset of distress. In the very young this distance is short; but with the years it gradually increases, until a maximum is reached in the heyday of early manhood. From that period onwards a gradual decrease sets in, the distance ever diminishing, till old age is reached. There is no standard. Some people are remarkably long-winded; others, equally healthy, are short-winded.

The Purpose of Distress in Effort

The purpose of the mechanism which thus limits for each of us the amount of effort we may undertake can fairly be described as protective; and it is well to keep this point of view in mind. It affords not only a measure of the heart's capacity, but also a rational line of treatment in heart failure. The mechanism is protective because of the universal dislike of distress. This dislike compels a man to cease effort before injury is done to his heart or other organs which are being strained. Little consideration, therefore, is required to understand how observation of the response to effort becomes the most important method of estimating the heart's efficiency. In eliciting this response we are calling into play

a mechanism which exists in every individual, healthy and diseased.

The Standard of the Heart's Efficiency

However, the doctor has to employ this method intelligently; and to do so he must undergo very careful training. The method is apparently so simple that everybody feels confident that he has recognised it, used it, and exhausted its possibilities. As a matter of fact, few people have given it anything like the amount of study it requires. In consequence its importance and the manner of its application are only partially understood.

The standard distance to be employed in determining whether or not an individual's response to effort is limited may at first seem a matter requiring definition. I have already referred to the fact that some people are long-distance runners, while others can run only a short distance. An amount of effort which would stop the latter may thus occasion the former no trouble. From this it can be seen that each has his own standard. It is therefore the duty of the doctor, by appropriate questions and tests, to obtain a knowledge of his patient's individual capacity, both in health and disease, before he attempts to determine the presence or the degree of heart failure. This information can be acquired only after much experience, as indeed may be understood when the various elements of the problem are taken into considera-

tion. Thus, some people with damaged hearts can walk long distances but cannot cycle, while others can cycle but are distressed by walking. Some people, again, suffer speedily on walking on a cold day or after a full meal. One man with mitral stenosis and auricular fibrillation followed his occupation as a mechanic, swinging his fore-hammer many hours, but had difficulty when walking up a hill.

CHAPTER V

THE LAW OF HEART FAILURE

The Response to Effort in Disease

The first sign of heart failure is a diminution of the power of this organ to respond to effort. The heart, as we have seen, holds in reserve a force which can be liberated to meet the demands of the body. The exhaustion of this force gives the key to heart failure. All the organs of the body, being dependent on the heart for an increased supply of blood during their activity, show an impaired efficiency when there is any failure to supply their requirements. Thus the first sign of trouble will be found not in the heart itself, but in the other organs. Their functions will be interfered with and they will give rise to symptoms.

The Nature and Mechanism of the Early Signs of Heart Failure

Bodily exertion is the most effective way of calling upon the whole reserve force of the heart. Exertion can be carried on at the command of the will; it employs great masses of muscles, and these require a great increase in their blood-supply during effort.

While during exertion the muscles demand and receive an increased supply of blood, the other organs of the body have their blood-supply impaired. Their functions are thus not properly executed. Most of the organs afford no sign of impaired function; there are, however, organs which are extremely sensitive to a diminished or depraved supply of blood.

The failure of blood-supply occurs in two ways: (1) by an impairment in quality; and (2) by a deficiency in quantity. During continuous effort in health and in disease a time comes when the output of the heart diminishes so that the organs do not receive a supply of blood suited to their requirements. This diminished circulation through the cleansing organs results in an accumulation of waste products in the blood.

The consequences of this accumulation react upon all the organs, but there are two in particular in which symptoms of a striking and easily recognised kind are produced. The symptoms are so characteristic that we must set them down at once. They are: (1) breathlessness and its associated phenomena, in consequence of the stimulation of the respiratory reflex; (2) pain and its associated phenomena in consequence of exhaustion of the heart muscle. Both may not be present in the one individual, but one or other is always present, however slight the degree of heart failure may be. In advanced conditions other organs and tissues may show signs—for example, dropsy and enlarged liver; but these are late manifestations and are not constant.

Respiratory Distress

The system most sensitive to an impaired blood-supply, or rather the system which most readily gives rise to sensations of distress, is the respiratory. Exactly how the distress is brought about need not concern us here; the point we are making is its association with heart failure. This is definite and beyond dispute. The impaired and impure blood-supply stimulates the respiratory reflex, so that the breathing is increased in rate. It is not, however, the rapid breathing which constitutes the distress, but that curious sensation called sometimes "air hunger," a feeling which compels the patient to take deep and rapid inspirations.

This breathlessness is the most common cause of distress in health and in disease. It is, in its quality, similar in both these states, as we have already suggested. Thus, a man in health runs a mile with ease, but suffers distress when this distance is exceeded; his heart becomes impaired, and then he may have the same symptoms of distress on running a few yards. With further impairment the breathlessness may come on during but a slight effort or even when he is at rest in bed. The reason for the breathlessness and distress remains the same in each instance. It is the failure of the heart to supply sufficient pure blood to the respiratory centre.

Pain

The next most common distressful sensation which compels cessation of effort is pain felt in some part of the chest, usually on the left side, and in the left arm. The inquiry into the production of pain in affections of the viscera has been a long and difficult one. Indeed the subject is still somewhat confused. A certain advance has, however, been made, which shows that there are but few conditions actually capable of producing pain. One of these is the contraction—or at least a form of contraction—of muscle. It is now many years since my attention was first drawn to the fact that some of the severest pains are associated with hollow muscular organs. For instance the uterus, the bowel, the ureter, and the gall duct. At a later period the study of heart affections revealed the fact that pain was evoked on occasions when effort was continued after exhaustion had taken place. This pain bore a relationship to the heart.

There is now sufficient evidence to show that, while the peristalsis or contraction of a hollow muscle may in many cases produce no sensation, contractions of a certain strength do produce pain. An analogy, too, exists between the production of this pain and that occurring in the skeletal muscles, for we find that, while the muscles usually contract painlessly, yet certain forms of contraction are very painful; for example, cramp. Moreover, if skeletal muscles

be forced to contract when they are exhausted, or—an even more severe test—when they are deprived of sufficient blood-supply, then pain of so severe a character may arise that effort will have to be abandoned.

We find the same kind of pain arising in connection with exhaustion of the heart. Conditions which enfeeble the heart muscle or impair its blood-supply favour the occurrence of pain when effort is made; and this pain is frequently of such severity as to cause a cessation of effort. The mechanism by which this effect is brought about is present in every individual, and the effect may be produced in the healthy as well as in those with impaired hearts. On several occasions I have noted its production in healthy people who had been exposed to great exertion for a long time; its production in damaged hearts is well known. From a study of a large number of cases, I conclude that pain arises from fatigue of the heart muscle, and is more easily produced when there is a deficient supply of blood to the muscle, as occurs in cases of disease of the coronary arteries. Its appearance in healthy people is due to fatigue from over-exertion. The pain may arise also in people whose hearts suffer, in company with other organs, from some general cause, as, for example, the poisoning from infection. I have dealt more fully elsewhere with the evidence which leads to this view of pain; I refer again to the matter in the chapter on "The Neurotic Heart."

The Mechanism by which Pain is produced

The following seems to be the mechanism by which pain and allied phenomena are produced. When a muscle is exercised beyond its power, exhaustion occurs; this acts by stimulating the nerve ends. These nerve ends convey to the central nervous system a stimulus which affects certain centres of which that giving rise to pain is one. The distress thus produced causes a cessation of the effort. The stimulation may affect not only the pain centres, but also other centres in the neighbourhood, such as the cells of nerves supplying certain muscles. In this latter instance the muscles contract, and thus we frequently find a sense of constriction as well as of pain.

The Sense of Constriction

There is, thus, a relation between the sense of tightness and the pain. In many of my cases of angina pectoris I have observed that the first consciousness of limitation of the field of response to effort is this weight or oppression or tightness across the top of the chest. The production of pain comes later. In some people effort produces first a sense of constriction; if the effort is persisted in pain is induced. It is for that reason that I lean to the view that the compression of the chest is due to a reflex contraction of the intercostal muscles. The sensation sometimes goes so far as to cause a feeling that the

chest is being gripped in a vice; this feeling resembles that accompanying the contraction of the intercostal muscles in pleurodynia, and also the hardening and contraction of the muscles of the abdominal wall in attacks of renal and gall-stone colic.

It would seem, therefore, that, while the air hunger or breathlessness is produced by the defective supply of blood to the respiratory centre, the pain and sensation of constriction of the chest are produced by a defective supply of blood to the heart muscle, and therefore due to exhaustion of the heart muscle.

The Relation of Symptoms to the Disease

The question often arises: "Why do many people suffer from the same pathological state, and yet have symptoms so diverse?" We know that many people die of heart failure without having suffered any pain. From what I have said already the reader will be able to understand why I believe that this is to be explained by a reference to the sensation of distress which is first called into play. A defective supply of blood to the respiratory centre may cause such distress in the shape of breathlessness that effort ceases before the defective supply affects the heart itself. In the same way pain may be the first sign of an exhausted heart in those instances in which the blood-supply to the heart muscle is markedly diminished. This view is supported by what happens in some people who develop

auricular fibrillation. In the great majority of people with this complaint breathlessness is the dominant symptom. It is produced by effort. Pain is much less frequent, and attacks of angina pectoris rarely occur. Patients who suffer from angina pectoris, and are compelled to abandon effort because of pain, sometimes develop auricular fibrillation. When this happens it is no longer pain which pulls them up, but breathlessness. Thus a man who could usually walk 200 yards before he was pulled up by pain developed auricular fibrillation, and was compelled to stop after 100 yards because of breathlessness. He had no more attacks of pain.

Exhaustion

Inquiry into sensations which compel cessation of effort discloses the fact that there are several of these. Exhaustion is perhaps the next most important, though it is not directly cardiac in origin. It is the sensation which compels people in indifferent health to cease walking or exerting themselves—a feeling which probably everyone has experienced from time to time, and which each individual would describe in slightly different words. There are a number of different kinds of exhaustion; these can be provoked in different ways and are associated with different diseases. Into that I do not propose to enter here, further than to state that exhaustion is not a sensation provoked by a failing heart responding to effort.

This is important, because a sense of exhaustion is commonly assumed to be an evidence of heart failure. As a matter of fact the feeling is produced by another process altogether, which has as its basis a modification of the vaso-motor system. The fact that exhaustion may occur in healthy people as a temporary phenomenon and under circumstances in which no strain is put on the heart—for example, standing in a hot and stuffy atmosphere—proves this. An inquiry into the whole subject is at present being undertaken. I may perhaps say that the sensation is found to be a symptom of much significance, and that it seems to vary with the nature of the disease present. It may, of course, arise readily in people with some actual heart trouble; this, however, is a different matter from saying that it is due to the heart trouble. Its presence should invariably be regarded as a reason for the examination of the patient for some cause of ill-health other than heart disease, to which—let me repeat—its relationship is only incidental.

Palpitation

The susceptibility of the heart to stimulation renders it liable to a variety of manifestations. There is scarcely a disease process, however trivial, or a modification of the activity of any organ, which is not reflected on some part of the cardio-vascular system; many of these disturbances affect some portion of the heart's own mechanism. It is on this account that we

frequently encounter disturbances in the action of the heart when the body is at rest. As is well known, mental effort or excitement will produce an attack of palpitation. Moreover experience shows that stimuli, from other organs, will produce the same phenomenon. When, therefore, we find a patient who complains of seemingly causeless attacks of palpitation, we should at once institute a search for the cause of these in some organ other than the heart; for it is rare, even in disease of the heart, for palpitation to arise without some extrinsic stimulus. I do not dispute, on the other hand, that certain states of the heart render it more liable to stimulation of this kind, and I mention in this connection febrile or toxic conditions due to general infections or absorption from diseases elsewhere.

Summary of Heart Failure

Evidences of heart failure are to be found not in the examination of the organ, but in the manner in which the circulation is maintained in the different organs of the body.

The symptoms of heart failure are to be found in the evidences of disturbed function of the organs of the body.

Such disturbed function is the outcome of an insufficient or depraved blood-supply.

While every organ of the body suffers when heart failure sets in, only a few organs produce recognisable signs.

Dropsy and enlarged liver are sometimes

spoken of as "cardinal signs of heart failure," but they occur only in heart failure from certain diseases and in an advanced stage of the condition. Most cases of heart failure do not show these signs at all, or show them only at a late period.

The organs which show the most distinctive signs of heart failure are the respiratory organs and the heart itself.

Although the signs are provoked by these organs, they are not directly due to them, but to their reflex action on the nervous system.

In the case of both these organs heart failure gives rise to distress; in the one case the distress is associated with breathlessness, in the other with pain of a definite kind in a definite locality.

There are thus two groups of symptoms which are provoked by heart failure, and which afford a standard for measuring the heart's capacity.

CHAPTER VI

HEART FAILURE WHEN AT REST

Heart failure may set in when the body is at rest; when, that is, there are no opportunities for testing the effect of effort except movements in bed. This occurs in the case of people who are forced to lie up on account of an accident, or a surgical operation, or pregnancy, or acute febrile infections.

The symptoms of failure in these states resemble those of certain forms of chronic heart disease when the patient is confined to bed; for example, advanced mitral stenosis or auricular fibrillation. It is in these latter cases that the best opportunity is afforded of studying the condition.

In the inquiry I carried out I observed cases of typhoid fever, of old people confined to bed after a fracture of the leg or after a surgical operation, of pregnant women, and of chronic disease of the heart.

Earliest Signs

The failure begins very insidiously, and unless carefully searched for it may easily escape notice until it is far advanced. The first symptom

which arrests attention is a slight increase in the rate of breathing. A slight acceleration of the pulse usually accompanies the increase in the breathing. This, however, may be masked by an acceleration of the pulse due to the complaint.

Dilatation of Right Heart

In certain cases, especially in typhoid fever or in chronic heart disease, there is an increase in the size of the right side of the heart, which is revealed by a slight extension of the transverse dullness, and by the appearance of pulsation in the epigastrium—a form of pulsation peculiar to the right heart.

In regard to these two signs, it must be noted that increase in the size of the right heart, of a mild degree, causes little increase of dullness to the right, because the right side of the heart is fixed by the attachments between the superior and inferior venæ cavæ. The increase pushes the whole heart to the left. In some cases, too, the greater part of the anterior surface of the heart is made up of the right auricle and ventricle. This can be verified by the character of the apex beat when it is present. When the apex is formed by the left ventricle there is an out-thrust during ventricular systolic (Fig. 3), and an indrawing during diastole. When it is formed by the right ventricle, the reverse takes place; an indrawing occurs during systole and an out-thrust during diastole, as in the latter part of Fig. 3.

In epigastric pulsation due to dilatation of the right ventricle, the movements are similar to those of the apex beat, when this is due to the

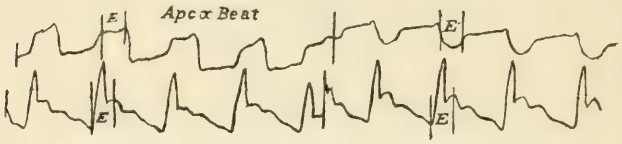

FIG. 3.—Simultaneous tracings of the heart movement (upper tracing), and of the radial pulse (lower tracing). The first part of the upper tracing was taken from the apex beat, immediately outside the nipple line, and shows a rise, due to the out-thrust of the apex, during the ventricular systole (space *E*). This out-thrust is characteristic of the movement of the left ventricle. The latter part of the upper tracing was taken immediately inside the nipple line, and shows a fall during the period of the ventricular systole (space *E*). This indrawing is characteristic of the movement of the right ventricle—receding from the chest wall as it empties.

right ventricle—an indrawing during systole and an out-thrust during diastole (Fig. 4). That point distinguishes epigastric pulsation due to

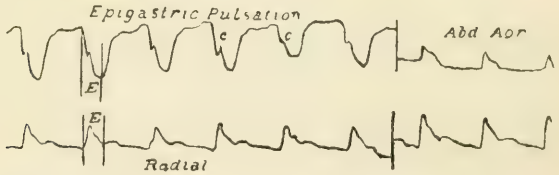

FIG. 4.—Tracings of the movements of the right ventricle in the epigastrium taken at the same time as the radial pulse. The epigastric pulsation shows an indrawing during systole (space *E*), due to the recession of the ventricle as it empties—resembling the latter part of the heart movement in Fig. 3. At the end of the tracing are two beats of the abdominal aorta, to show the difference between the epigastric pulsation of the right heart and that of the aorta.

the right ventricle from epigastric pulsation due to other causes; for example, the left ventricle, the abdominal aorta, or the liver—all of which

show an out-thrust during systole. It is to be noted that this increase of the right heart may take place with no perceptible modification of the heart sounds.

Œdema of the Lungs

The most instructive signs of early failure in bed, however, are the changes which take place at the bases of the lungs. The systematic examination of weakly people, confined to bed, reveals frequently a few fine crepitations at one or other base of the lung, behind. In the early stage of failure those crepitations disappear after the first deep inspiration. In many weakly people the condition goes no farther ; in those, however, in whom heart failure proceeds, the crepitations do not disappear during deep inspiration, but persist. In course of time an impairment of the percussion note may be detected, and the condition may go on till there is marked dullness of one or both bases. Disappearance of the crepitations and the breath sounds now occurs. A post-mortem examination of such cases shows the lungs to be sodden and airless at the base. Sometimes patches of catarrhal pneumonia are found in the substance of the œdematous parts of the lung.

With the increase of this passive congestion other symptoms appear. The breathing becomes more hurried ; the act of turning over in bed produces evident dyspnœa ; the pulse-rate increases ; if there be catarrhal pneumonia, the

temperature rises with its onset, and the patient becomes manifestly ill.

Method of Detecting the Onset of Pulmonary Stasis

It had been recognised that stasis of the lung tends to occur in cases of pregnancy complicated by mitral stenosis. Bearing this in mind, I extended my inquiry to include not only people with damaged hearts, but healthy women during pregnancy and people suffering from any form of debility. My object was to find out whether or not crepitations at the bases of the lungs are a sign of danger in pregnancy when they occur in a woman with heart disease. I examined and kept notes of women in the manner described in Chapter II, p. 12.

I found by this means that crepitations at the bases of the lung are far from being an uncommon sign. The method I adopted was as follows : I told the patient to stay in bed on a particular day. When I visited her I observed which side she lay on, and before attempting any further examination made her sit up. While I auscultated the base of the lung on the side on which she had been lying, she took a deep breath. In most cases no crepitations were heard ; in others fine crepitations were heard which disappeared after the first deep inspiration.

Such people went on to full time, were confined, and suffered from no heart trouble. Even those in whom the crepitations were more persistent

did not have any cardiac trouble. In all of them the crepitations disappeared during the strain of labour, though many showed crepitations at the base a day or two after the delivery. No bad consequences followed. I noted, however, that the crepitations all disappeared on taking a few deep breaths.

In those women who had mitral stenosis or auricular fibrillation, and who gave trouble on account of their hearts, the crepitations did not disappear on deep breathing, but persisted. In some the lungs became dull to percussion from an increase of œdema. The œdema occasionally grew worse till the breathing became embarrassed ; the œdema after confinement was often more pronounced than before it.

While, therefore, crepitations at the base, in a healthy woman, were shown to be a sign of no serious significance, they gave an indication of danger when the pregnancy was complicated by heart disease.

The Mechanism of Right Heart Failure

While in some people heart failure can take place and proceed far without demonstrable physical sign, in others œdema of the legs is a prominent feature. It is not quite clear why there should be this difference ; personally I reasoned that dropsy is in some way or other associated with a diminished force of the left ventricle, and is, in all probability, due to a diminished *vis-a-tergo*. This view was strength-

ened by the observation that true cardiac dropsy is always associated with a dilated left ventricle.

I had frequently been confronted with cases of dropsy in which an abnormal sign such as a systolic murmur was present. I assumed that the dropsy was due to a failing heart. But I found that some of these cases showed no enlargement of the heart, and in the course of time it became apparent that their dropsy was not of cardiac origin. I therefore came to the conclusion that in all cases of *cardiac* dropsy the left ventricle is dilated, and so is not able to overcome the peripheral resistance. What other factors there are, beyond this, I do not know ; nevertheless the recognition of an association, even if it be not cause and effect, is of great clinical value.

Reasoning from these facts, I looked upon the crepitations at the base of the lungs as being due to œdema, occasioned by a diminution of the force of the right ventricle. The association of œdema of the lungs with the demonstrable evidence of dilated right heart, already described, supported this view. I then set about to ascertain why it was that heart failure should set in while the body remained at rest, and why it should take this particular form.

When one reviews the factors which favour the flow of blood through the lungs, two of these stand out prominently :

(1) The force of the right ventricle ;

(2) The movements of respiration.

The force of the right ventricle is the main factor, but the free movement of the chest wall

undoubtedly helps. When a heart fails the patient sits up and breathes in a laboured fashion. When a person lies down pressure is exerted upon the ribs so that their movements are restricted.

With a good right ventricle there is no difficulty in maintaining the circulation through the lungs. With a weakened one, or with an embarrassed pulmonary circulation, the case is altered and the circulation is not maintained efficiently. The blood tends to stagnate in that part of the lung which is passive during respiration. This probably is the reason why the first signs of right-heart failure are the crepitations at the base of the lung on the side on which the patient lies. I have repeatedly seen all signs of stasis disappear when the patient was made to sit up and when, at times, he breathed deeply.

The fact that mitral stenosis hinders the flow of blood through the lungs, and therefore embarrasses the right heart, accounts also for the fact that œdema of the lungs is an early sign of the failure of the right ventricle. Having assured myself of this, I put the question why it should be specially serious in pregnancy. In order to find an answer, I made a careful study of certain changes which take place in a normal pregnancy. It is perfectly evident that in the pregnant state, towards the later months, the heart is embarrassed in healthy women; for the woman is not capable of so much exertion as she was accustomed to undertake, and becomes breathless during efforts which at other times would not have inconvenienced her.

The Source of Danger in Pregnancy with Mitral Stenosis

The embarrassment of the circulation which occurs during pregnancy was described in Chapter II, where it was shown that the growth of the uterine tumour interferes with the movements of respiration, by widening the thoracic cavity, preventing the descent of the diaphragm and displacing the heart. The result was seen to be a limitation of the heart's power to respond to effort, and a tendency to stasis at the bases of the lung.

In some cases of mitral stenosis the blood is obstructed at the mitral orifice, the left auricle becomes full and embarrassed, engorgement of the lungs occur, and the right heart has a difficulty in forcing the blood through the lungs. In mitral stenosis where this state of affairs is already present the additional embarrassment caused by pregnancy places the woman in a precarious state. This point, however, will be more fully discussed under mitral stenosis.

The Back-pressure Theory of Heart Failure

The explanation given of the mechanism of heart failure in mitral stenosis has led to the idea that this is the usual manner of heart failure, and in consequence there has arisen what is called the back-pressure theory. Although, as

I have pointed out, there is some truth in this theory in certain cases, yet in the majority it gives a wrong impression. With the discovery of murmurs, and their relation to valves found damaged at the post-mortem examination, there arose the idea that the embarrassment of the circulation in heart failure was due to the incompetency of the valves. This idea seemed to be strengthened by the dropsy and œdema of the lungs which were also frequently present. It was taught that where any difficulty occurred at the aortic orifice, the left ventricle would be embarrassed, and dilatation would follow. As a result of this dilatation, the mitral valve would no longer be able to close the orifice, so that during systole the blood would flow back into the left auricle. This in time would, in its turn, become distended, and so lead to interference with the pulmonary circulation and to engorgement of the lungs. The right ventricle thus must be embarrassed also, and would sooner or later dilate so that the tricuspid valves would be rendered incompetent and cause distension of the right auricle, and then engorgement of the veins of the body, enlargement of the liver, and dropsy. By some people to-day a tricuspid murmur is looked upon as a sign of complete heart failure.

It was on this plausible reasoning that the back-pressure theory of heart failure was built up ; and it is because of this reasoning that murmurs are looked upon as being so ominous.

Seeing that this belief is so widely accepted by

authorities and teachers, one might reasonably expect that, in the vast literature dealing with affections of the heart, there would be recorded at least one case showing the progress of heart failure after this fashion. I have searched diligently, but I have not found the record of one single case which could support the idea. Moreover, I have, time and again, asked its supporters to give me a record from their own experiences of heart failure arising in this way. None of them have done so. So long a period has elapsed since I first called attention to this misconception, that plenty of time has been afforded physicians to produce evidence in support of it. They have failed to produce any evidence.

The reason why the erroneous view is held is that there are present in certain forms of heart failure marked evidences of engorgement; for example, congestion of the veins, lividity of the face, enlarged liver, and dropsy. These evidences are invariably pointed to as proofs of "back-pressure." But if one watches the onset of those symptoms, one will be able to perceive that they arise in another way.

The Mechanism of Heart Failure

While watching for a period of years the changes which gradually occurred in my patients with mitral disease following rheumatic fever, and in elderly people, I was struck with the suddenness of the onset of heart failure in these subjects. It came accompanied by great breath-

lessness, lividity of face, enlargement and pulsation of the liver, and dropsy. During my observations I had taken notes of the various signs; on comparing the signs before and after the onset of heart failure, I found some remarkable differences. Thus, before the onset of heart failure the organ would be perfectly regular, while after the onset it would be irregular. The irregularity, too, was of a peculiar and distinctive kind.

Moreover, while before the onset of heart failure there were evidences of auricular activity, these disappeared with the onset. (These evidences of auricular activity were movements in the jugular veins or the liver due to contraction of the auricle, as in Fig. 2, and, in some cases, presystolic mitral or tricuspid murmurs. These all disappeared on the onset of heart failure.) These changes were afterwards found to be due to the occurrence of fibrillation of the auricle.

In some of my cases the auricular fibrillation lasted but a few hours or a few days. In a few, the patient, immediately after the onset of the fibrillation, became breathless. Some hours later the face would become livid, the heart dilate, and the liver enlarge. The heart would sometimes suddenly revert to its normal, regular rhythm—the auricle beating again in a normal fashion. In these instances the patient at once experienced a great sense of relief, and in a few hours the face had regained its usual colour, and the heart and liver diminished in size. Effort

would then be undertaken without breathlessness.

While I do not profess to comprehend the whole of the mechanism of heart failure, it is manifest to me that this sequence represents a very different picture from that which could be presented by "back-pressure." So far as I can understand it, the real process seems to be as follows: In cases of auricular fibrillation with heart failure the rate of the heart becomes increased, often greatly so, while the beats are

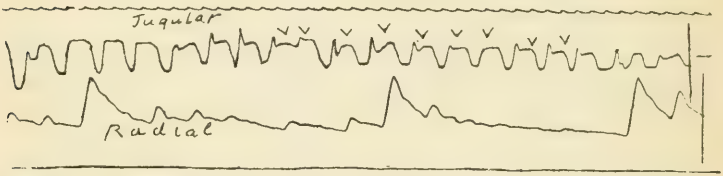

FIG. 5.—Characteristic irregularity appearing at the onset of auricular fibrillation. The upper tracing is from the jugular vein and shows large waves (*v*) with each systole of the right ventricle. The lower tracing is from the radial, and shows in addition to a marked irregularity that the beats are sometimes so small that the instrument fails to record them.

weak and ineffective, and the radial pulse is small and sometimes scarcely perceptible (Fig. 5). The chief force which sends the blood through the systemic circulation to the right auricle is the left ventricle. When we find the ventricular beats small and ineffective, it is a reasonable surmise that such concomitant symptoms and signs as dropsy, enlarged liver, and lividity are due to the inability of this chamber of the heart to force the blood through the peripheral vascular system.

The inability of the left ventricle to expel its

contents leads naturally to an accumulation of the blood in its cavity. Thus dilatation may occur. It is not, however, the accumulation of the blood alone which causes dilatation. In many hearts, in which fibrillation of the auricle is present, there is no dilatation. Dilatation indicates that the wall of the ventricle is damaged or impaired ; its presence should always lead to consideration of the integrity of the heart muscle.

The importance of auricular fibrillation as a factor in the production of heart failure should be recognised. In my observations I found that about 90 per cent. of cases of heart failure with dropsy and enlarged liver were cases of auricular fibrillation. Moreover, in the great majority of cases of "heart-strain"—i.e. where heart failure with breathlessness has suddenly set in while the individual was making a violent or prolonged effort—the failure is due to the sudden onset of auricular fibrillation.

Auricular fibrillation is often attended by little or no impairment of the cardiac efficiency. In such cases the heart muscle is good, and the rate of the heart is not markedly increased.

There are other, but rarer, conditions which induce heart failure, but as they seldom, if ever, occur in connection with pregnancy they need not be discussed here.

While "back-pressure" fails to account for the symptoms of heart failure, it is nevertheless a factor in some cases. In mitral stenosis there can be no doubt that the left auricle is often

embarrassed, and in some cases the pulmonary circulation shares in this disability. The right ventricle hypertrophies to overcome the difficulty in the pulmonary circulation. This point is of importance as it throws some light on the danger of pregnancy in mitral stenosis.

CHAPTER VII

MURMURS

Misconception of the Significance of Murmurs

I recognise at the outset that in dealing with murmurs I have to overcome a great number of erroneous notions held by the medical profession with conviction. The idea that a perfectly sound heart may show a murmur is so surprising to many doctors that they cannot accept it. Their view seems to be that if murmurs are not organic they are functional and imply debility or some other condition of weakness. That a murmur may be as physiological as a pure sound is altogether outside the limits of this faith. I know that some doctors are ready to admit that some murmurs may be without significance ; but nowhere have I found a description which makes clear the exact difference between murmurs of serious significance and those which are innocent. It is on this ground that teachers have failed to enlighten their students. They hedge so much in their descriptions that their pupils go forth with vague ideas and consider it wiser and safer when in practice to view all murmurs with suspicion. I speak of this from a wide

experience derived not only by seeing individuals to whose murmurs an unnecessarily grave significance has been attached, but also by questioning doctors as to their views.

In no field of medicine, so far as I know, has this attitude towards murmurs proved so disastrous as in pregnancy. The detection of an innocent murmur has often supplied a reason for forbidding pregnancy or even marriage. Indeed when I ponder on this subject I am sometimes inclined to think that the discovery of auscultation has actually done more harm than good to the practice of medicine.

It is necessary, if we are to make any progress at all, to understand wherein lies the danger associated with murmurs. Most murmurs seem to arise at the valve orifices of the heart. It has been demonstrated that certain damaged valves give rise to abnormal sounds, and it is evident from the appearance of the valves post mortem that the flow of blood must have been hindered on passing through the orifice or that the valves, being incompetent, have allowed blood to escape backwards. In this way the work of the heart muscle has been hampered. An additional burden being thus thrown upon his heart, a man leading his usual life puts a greater strain on this organ than it is fit to bear. Thus it is only a matter of time until the muscle becomes exhausted and heart failure sets in. But there are murmurs which do not indicate damaged valves, or valves damaged to such a degree as to cause embarrassment to the heart, and it is

necessary to distinguish between these murmurs and those which indicate valve damage which embarrasses the heart.

Physiological Murmurs

Physiological murmurs are always systolic in time, and it is impossible to tell the origin of the greater number of them. They may be loudest at the apex or at the base, or at mid-sternum; they may vary with respiration or posture; sometimes they are heard when the patient is lying and disappear when he stands up; again, they may be present on standing and disappear on lying down. Much time has been spent in speculation on their causes, and fanciful names have been bestowed upon them. From the point of view of the heart's efficiency they are negligible. They indicate neither disease nor impairment, and do not foreshadow the oncoming of any heart trouble. I make this statement as wide a one as possible, for I have watched great numbers of people at all ages—the young growing into manhood and womanhood, leading strenuous lives, and in the case of the women bearing children—yet in no case have I seen any harm result from the fact that their doctor ignored these systolic murmurs. Moreover, there is not to be found in the whole literature of medicine, so far as I am aware—and I have diligently sought for such evidence—a record of a single case of an individual, man or woman, who suffered from heart failure of which a systolic murmur was the only symptom.

Functional Murmurs

Functional murmurs are usually assumed to be due to dilatation of the heart; the orifices of the valves are supposed to be rendered so wide that the valves fail to close them. This is not entirely correct, for while it is true that we may find a murmur with dilatation of the heart, we may often find great dilatation without a murmur. Further, murmurs come and go when there is no perceptible change in the size of the heart; they may, for example, appear only when the organ is excited or when the patient stands up, and may disappear when the heart quiets down or the individual is at rest. Again, murmurs which we find when the heart is acting quietly with the body at rest may disappear on exertion or excitement.

From this it can be inferred that there is some special mechanism, apart from that involved in the dilatation of auricle and ventricle as a whole, which is capable of producing a murmur. This obscure mechanism varies in its efficiency according to circumstances which we are still unable to appreciate. The importance of this is most clearly seen in connection with the hearts of young people, in whom it is probable that the organ has a peculiar power, which is gradually lost in later life, of adapting itself. It must be remembered also that free regurgitation can take place at the mitral and tricuspid orifices without producing any murmurs. We frequently get evidence of marked tricuspid regurgitation in the

veins of the neck and in the liver, yet cannot detect a tricuspid murmur when we come to listen to the patient's heart.

Systolic Murmurs and Heart Failure

These systolic murmurs, whether functional or organic, are thus supposed to be originated by an escape of blood back from the contracting chamber. This is called regurgitation and has, as we have seen, been assumed to be the chief factor in the production of heart failure. From the theoretical study of it has arisen the "back-pressure" view of heart failure.

There are, however, no accurate data regarding the quantity of regurgitant blood necessary to produce a murmur; and so we are forced to estimate this amount by indirect methods. I have been able to keep under observation, for twenty or thirty years, individuals with loud, rasping, systolic, mitral murmurs, who gave a history of rheumatic fever, and so permitted me to conclude that the murmurs were due to some damage of the mitral valves. These patients never suffered from heart failure. I venture, therefore, the conclusion that, in them, the leak at the valve was small and the damage slight. One may thus say that within certain limits, and in respect of mitral murmurs due to damaged valves, the smaller the leak the louder will be the murmur. In the case of functional murmurs, the leak, if there be a leak, is so slight that it never, except in cases where there exists grave

damage to the heart muscle, embarrasses the auricles in their work. The small quantity of blood thrown back does not alter appreciably the size of the auricles, for we often find murmurs in hearts of the normal size.

The Differentiation of Functional from Organic Murmurs

It is not always easy to distinguish between functional and organic murmurs. As a rule functional murmurs are systolic in time and are heard with equal clearness over different parts of the heart. They are usually soft and blowing. But these characteristics do not distinguish them from some organic murmurs. Indeed, though much has been written, and though many attempts at exact differentiation have been made, it remains true that even the most seemingly characteristic organic murmurs resemble functional murmurs so closely that it is not possible in all cases to arrive at certainty. A very rough murmur, especially if accompanied by a purring tremor or a musical note, is indicative of a valve lesion. In the early stages of an endocarditis, however, the murmur is usually soft and blowing, and so presents "functional" characteristics.

To distinguish between these two murmurs the whole circumstances of the case must be considered. In the first place, it is necessary to bear in mind that a murmur may be a normal event. When we find a systolic murmur occurring in an individual in robust health who reveals no other

cardiac sign, such as a thrill or an increase in the size of the heart, we may consider the murmur as indicative neither of impairment nor of disease. When we find a murmur occurring in a debilitated person, we should avoid arriving at any conclusion until a search has been made for conditions which may account for the debility. Examples of such conditions are anæmia, imperfect nourishment, insufficient rest, and disease of other organs. It is of great importance, too, to place in their true perspective murmurs occurring in febrile cases, or after the subsidence of an acute febrile attack such as rheumatic fever. The question in these instances is whether the heart has been affected by the disease process. It is always necessary to bear in mind that the raised temperature or the toxins of the illness may produce changes in the heart's action as opposed to changes in its structure, and so may give rise to murmurs and even dilatation which have no organic basis in the strict sense. During the febrile stage of an acute illness, therefore, judgment should be suspended. It should remain in suspense until definite signs of an organic lesion are forthcoming. After a febrile attack a murmur which has arisen during the attack may be observed to persist, or again a murmur may arise at this time *de novo*. In these cases the other signs and symptoms present must be observed and weighed duly before conclusions are reached. Thus if, after the subsidence of the fever, the rate of the heart becomes slow and the respiratory type of irregularity develops, it

may be assumed that the heart has escaped infection and that the murmur is functional. If, on the other hand, the rate is persistently increased, fear may be justified that the heart has become infected, and that active changes are going on.

The Significance of Systolic Murmurs

Since it is true that individuals with systolic murmurs may be in perfect health, may be leading strenuous lives, and may never show any sign of heart failure, we are entitled to conclude that these murmurs may be a physiological and normal sign, and indicate no impairment of the organ's efficiency, nor foreshadow the oncoming of its failure.

I wish to insist upon this, and I base my insistence on the fact which I have already stated, that I have watched numbers of healthy young people, who exhibited such murmurs, grow up into manhood and womanhood, lead healthy and vigorous lives, and never show the slightest sign of heart failure. We know so little of the causation of murmurs that we have small justification indeed for treating them as abnormalities. Even if they are due to regurgitation, it is quite conceivable that this regurgitation may be consistent with a good functioning heart. I have already pointed out that the fear of regurgitation has attained the dimensions of a bogy in the medical mind ; further, I have pointed out that, even in cases of organic disease, the amount of regurgita-

tion necessary to produce a murmur is probably very slight. Again, functional murmurs may be present in debilitated individuals; in these cases the murmur is not necessarily an indication of disease or impairment of the heart. Nor is the valve incompetency which its presence indicates the cause of the debility. Manifestly, a murmur is thus but one of many symptoms which an individual may exhibit. Sound diagnosis demands therefore a search for other evidences and signs which may throw light on the fundamental cause of the debility. I do not deny, in saying this, that a systolic murmur may be present in cases of grave heart failure, but I do urge that in these, as in all other cases, it is but an associated symptom. The valvular incompetence is not the cause of the heart failure; it is merely one of the changes which have arisen in consequence of impaired functional efficiency of the heart muscle.

The Significance of Organic Murmurs

I feel that I cannot emphasise this truth sufficiently. That organic murmurs have a varied significance is true, and that they are associated with heart failure is not in dispute; I am nevertheless convinced that the valve lesions which they indicate are in most instances not the cause of the failure. We cannot afford to forget in this respect that individuals with murmurs due to damaged valves caused by some infective disease, such as rheumatic fever, may

lead vigorous lives and be engaged in strenuous occupations, and may never show any sign of heart failure.

We conclude, then, that valve lesions in themselves may not necessarily be of serious significance. As, however, murmurs are often associated with heart failure, the presence of organic murmurs would give rise in our minds to the following considerations :

(1) Whether we have here an indication that the heart has been invaded by a disease process ;

(2) Whether the damage done by the disease process is stationary or is slowly progressive ;

(3) Whether the disease process has affected and impaired the heart muscle as well as the endocardium ; and

(4) Whether the damage to the valve is such that it obstructs the work of the heart and so embarrasses the muscle and impairs its efficiency.

In acute illnesses, as already suggested, it will not be possible to answer these questions, for there the immediate consideration is the infection itself. In chronic cases—that is to say, cases described as chronic valvular disease—it is absolutely necessary that these questions should be considered and answered if a rational conclusion is to be reached. The method of obtaining the knowledge necessary to answer them will be described in connection with the different valve lesions.

To sum up : estimation of the significance of murmurs, as of all other signs, should be based not on the murmur itself, but on the functional

efficiency of the heart, and on the presence or absence of additional symptoms of cardiac mischief (size, rate, and rhythm). If we find in a heart of normal size and rhythm (or with the respiratory type of irregularity) a systolic murmur unaccompanied by evidence of cardiac inefficiency, we may conclude that this heart is perfectly sound. If there be present evidence of weakness, or of other abnormal conditions, then the opinion we reach should be based on these conditions, and not on the murmur.

CHAPTER VIII

MITRAL REGURGITATION

Nature of the Valve Changes and Character of the Murmur

Mitral regurgitation may be the result of a damaged valve, or of dilatation of the auriculo-ventricular orifice from the giving way of the muscles supporting the valve. The murmur of mitral regurgitation is systolic in time; it is loudest at the apex. It may be soft and blowing and of little intensity. Sometimes in these cases it is heard over a very limited area; in other instances it is propagated into the axilla; again, it may be rough and loud, and may be heard over the whole heart and round at the back of the chest. It is not always possible to tell whether this murmur is due to dilatation of the auriculo-ventricular orifice or to damage of the valve. The rough, loud type, with an accompanying thrill, is always a sign of a damaged valve.

Mitral Regurgitation and the Heart Muscle

When the muscle of the heart is unimpaired, little or no bad effect follows incompetence of the mitral valve. Even in those cases in which the

regurgitation is due to "functional" dilatation of the orifice, the contractile power of the muscle may maintain a good and efficient circulation and the patients enjoy perfect health. The really serious trouble in connection with mitral regurgitation arises when the myocardium is damaged; the regurgitation may then add to the embarrassment. Yet it should be remembered that until the muscle actually gives way few symptoms of heart failure may be produced.

Dilatation is generally looked upon as the result of mitral regurgitation, the idea being that back-pressure ultimately causes the walls of the right heart to yield. This is not quite correct, for long before there is any back-pressure we may find evidence of a dilated right heart. Thus, if during one of the slight attacks of heart failure which are likely to occur after over-exertion we investigate the condition of a heart—in which the valves have been damaged by rheumatic endocarditis—we may find the organ slightly dilated, the right ventricle in front and the left ventricle pushed to the left, behind the lung. The apex beat in such a case is caused by the right ventricle, as is shown by the occurrence of an indrawing of the beat during systole. After a few days' rest and treatment, this right heart may retreat; the apex beat will then be caused by the left ventricle, and the beat will present the normal character, i.e. an out-thrust during systole (see Fig. 3). In cases of this type there is no evidence whatever of pulmonary engorgement, and back-pressure.

Damage to the valve is most commonly the result of rheumatic endocarditis, and, as we have seen, the process is rarely limited to the endocardium, but invades likewise the myocardium. Septic endocarditis also may damage the valve. In all cases of mitral stenosis there is mitral regurgitation; but the amount of the regurgitation is never so marked as to be the serious factor in the case.

Regurgitation occurs through a mitral orifice, of which the valve is uninjured, in the later stages of many affections—for example, conditions which produce exhaustion of the heart muscle and more especially renal disease and cardiosclerosis. Here the mischief is brought about by the failure of the muscle to support the orifice; regurgitation may in such cases furnish one sign of a final and fatal exhaustion of the heart muscle. In fact, as Graham Steell says in a description of the post-mortem findings, "The change in the valves is altogether inadequate to explain the evidently free regurgitation that occurred during life, and the disastrous dilatation of the heart. The muscle-failure factor, it may be presumed, was the essential one."

It will thus be seen that the symptoms produced by mitral incompetence are only grave when there is also muscle failure. In this latter event we shall encounter those symptoms of heart failure in general which we have already discussed, and the earliest of which is usually breathlessness on exertion. When dropsy sets in, auricular fibrillation (q.v.) is commonly present.

Mitral Regurgitation with Auricular Fibrillation

The necessity of assessing the significance of mitral regurgitation in terms of the presence of signs of damage to the muscle becomes very evident when we examine the literature of this subject and note how great is the confusion of mind which inevitably results from an imperfect understanding of the nature of symptoms.

Angus Macdonald in his book quotes the cases of two women with mitral insufficiency who passed safely through repeated pregnancies (Cases XV and XVI). He then quotes the cases of two other women (XVIII and XIX) with mitral insufficiency who died from heart failure shortly after delivery. The one sign common to these four cases was a mitral systolic murmur. When, however, the other recorded signs are studied, a striking difference between the two groups is found. In the first group—those who did well—the response to effort was good; in the second—those who died—breathlessness on effort was noted and is clearly described. In one of those who died (Case XIX), the pulse was "rapid and irregular," an almost sure indication that the patient suffered from auricular fibrillation—a matter of far more importance than the murmur. The post-mortem examination showed not only a damaged mitral valve, but a damaged myocardium.

Mitral Regurgitation and Pregnancy

The detection of a mitral systolic murmur in a woman who is pregnant or who may become pregnant should cause the physician to consider the following points :

(1) The response to effort ;
(2) The size of the heart ;
(3) The rhythm of the heart.

If the response to effort is good and the heart is not increased in size, then the murmur requires no further consideration, as in all likelihood it is physiological.

If there be an increase in the size of the heart, but no diminution in the response to effort, and if the circulation is well maintained, then pregnancy may be allowed, even if there be a history of rheumatic fever.

If the size of the heart is increased and the response to effort is limited, then the case requires careful consideration. It must be determined whether the limitations are or are not due to a temporary cause and whether or not the heart muscle has been damaged. If no doubt remains that the heart muscle is damaged, then pregnancy should not be permitted.

If there is present an irregularity of the rhythm of the heart, the nature of this must be carefully investigated. Should the irregularity prove to be of the youthful or respiratory type, then there will be no danger if pregnancy is incurred. If, again, it is due to extra-systoles, no fear need be occasioned by their presence, for these of them-

selves afford no clue to the state of the heart's efficiency. In such instances opinion must be based on the presence or absence of other signs (size of the heart, response to effort).

Finally, if the irregularity is due to auricular fibrillation, opinion should be based on the circumstances to be described under that heading. The whole subject of irregularity is dealt with in a later chapter.

CHAPTER IX

MITRAL STENOSIS

The heart affection which most frequently causes danger in pregnant women is that associated with mitral stenosis following rheumatic fever. Although this has long been recognised, yet we find some authorities to-day declaring that there is little danger. The reason for the diversity of opinion which prevails is that, in many cases, experience is limited. For it is a fact that there are forms of this complaint which are attended with no danger, and there are others which imperil the life of the woman. It is necessary, therefore, if we are to distinguish between the different forms, to look at mitral stenosis far more minutely than has usually been done.

The Origin and Progress of the Cicatrising of the Mitral Valves

Mitral stenosis is, perhaps, the most common of valvular defects with which heart failure is associated. It arises generally in consequence of rheumatic endocarditis, though it may be found in people with no rheumatic history ; in these instances a previous history of erysipelas

or some other febrile complaint may give a possible clue to its origin.

The condition is never recognised during the acute illness which induces it, for the reason that stenosis does not occur till the cicatrising process which follows the inflammation narrows the orifice. Thus on account of its origin in scar-formation, the lesion is often a progressive one. The stenosis may, however, remain moderate in amount, and offer so little embarrassment to the heart that patients may reach extreme old age with no heart failure. Usually the cicatrising process goes on with varying rapidity until in some cases the mitral orifice is reduced to a mere slit, and the valves resemble a thickened calcareous diaphragm. It is thus always important to bear the probably progressive nature of the lesion in mind. We have here the secret of the varying changes in the symptoms.

The Accompanying Changes in the Heart Muscle

It should also be borne in mind that a similar cicatrising process may be going on in the muscle, causing contraction of the chordæ tendineæ, impairing at other places the functional activity of the heart muscle, and affecting the auriculo-ventricular bundle and depressing its conductivity or producing those conditions which lead to auricular fibrillation. Such changes profoundly affect the efficiency of the heart and modify the nature of its rhythm.

Cause of Heart Failure

From this it can readily be understood that the manner in which heart failure is brought about in mitral stenosis is somewhat complicated. In some patients embarrassment may not ensue until the narrowing of the orifice has become extreme. In others there may be a fatal issue while the narrowing is yet moderate. In these latter the muscle wall will invariably be found to have been damaged.

The Presystolic Murmur in Mitral Stenosis

The characteristic murmur present in mitral stenosis, the so-called presystolic murmur or auricular systolic murmur, is due to the passage of the blood through the narrowed mitral orifice. During the whole of ventricular diastole the blood is passing through the orifice, but until the contraction of the auricle forces it through with increased rapidity no sound is heard. With advancing cicatrisation, the murmur of mitral stenosis alters and presents peculiarities which have not hitherto been sufficiently appreciated. In the very early stages, some years before the appearance of a murmur, I have detected a slight presystolic thrill. After a time the thrill was accompanied by a murmur. This murmur precedes, or runs up to, and seems to terminate in the first sound ; it is audible over

a small area around the apex. At the beginning it may not always be present. This murmur is at first short in duration. As time goes on and the mitral orifice becomes narrow, it lengthens, beginning earlier in the diastole. It is of a crescendo character; rising in pitch till it ends in the first sound.

The Diastolic Murmur in Mitral Stenosis

With advancing stenosis of the orifice, another murmur makes its appearance. This one occurs immediately after the second sound, and is heard only in the immediate neighbourhood of the apex beat. At first it is very faint, and not very constant; but in time it increases in volume. This diastolic mitral murmur diminishes in intensity as it proceeds, differing thus (in its diminuendo character) from the presystolic murmur. Frequently during the diastole of the heart we can detect a continuous murmur, beginning loudly, falling away, then increasing in intensity. The first or diminuendo portion of such a murmur is the diastolic mitral murmur, the terminal crescendo portion is the presystolic. The cause of the diminuendo diastolic mitral murmur is the passage through the narrowed mitral orifice of the blood which has accumulated in the auricle during the ventricular systole. This passage begins as soon as the mitral valves open—that is, when the pressure in the ventricle falls below that in the auricle. The development of the murmurs is shown in Fig. 6.

The Appearance of Auricular Fibrillation

The next change in the character of these murmurs is the sudden disappearance of the presystolic crescendo murmur, the diastolic murmur persisting. Usually this occurs with the onset of grave symptoms of heart failure, the action becoming rapid and irregular. At other times

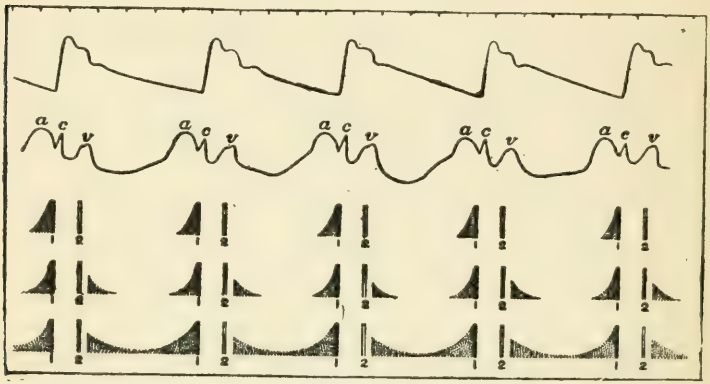

Fig. 6.—Tracings of the radial and jugular pulse with diagrammatic representation of the changes in the murmurs of mitral stenosis. The down strokes 1 and 2 represent the first and second sounds of the heart. The first indication of stenosis is the appearance of the murmur before the first sound, and on reference to the jugular tracing this murmur will be found to coincide in time with the wave *a*, due to the systole of the auricle. With an increase in the stenosis, a short diastolic murmur appears, immediately after the second sound, and this is represented in the middle diagram. A further increase in the stenosis, shown in the lowest diagram, leads to prolongation of the diastolic murmur, which in combination with the presystolic murmur fills up the whole interval between the second and first sounds. (For the next change see Fig. 7.)

the change takes place with no serious symptom, though the heart invariably becomes irregular. This irregularity is due to the fact that the rhythm of the organ no longer starts with an effective auricular systole. The auricle, on the contrary, ceases to contract as a whole, and passes into the condition of fibrillation (Fig. 7, see Chapter XII).

In those cases in which the heart's action is slow there is no difficulty in recognising the presence of the diastolic murmur and the absence of the presystolic. The diastolic murmur is, however, sometimes of greater length, and starts immediately after the second sound; in such cases, when the heart's action is rapid, it may fill up the whole diastolic pause, and might then be assumed to be presystolic. If the heart be carefully auscultated this error will be avoided, for the dias-

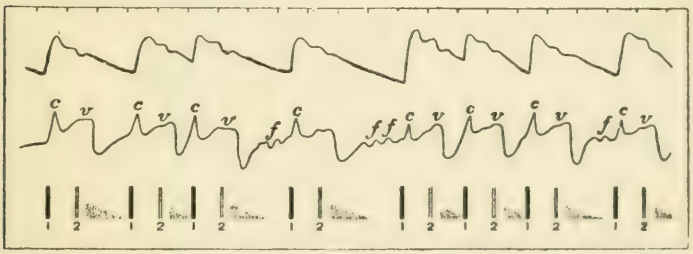

Fig. 7.—After the changes described in Fig. 6 the next change is a sudden alteration of the rhythm of the heart. In Fig. 6 the heart is quite regular; in Fig. 7 it is irregular. The wave *a*, which is a marked feature in the jugular tracing of Fig. 6, is absent in Fig. 7. The presystolic portion of the murmur is also absent in Fig. 7, this feature being best appreciated during the longer pauses of the irregularity. The disappearance of these evidences of auricular activity and the irregular action of the heart indicate the onset of auricular fibrillation. The waves *ff* are due to the fibrillating auricle.

tolic murmur is not crescendo in character. In auricular fibrillation long pauses may occur between two beats; and if this murmur be listened to then, it will be found that it stops short before the first sound, so that there is a silence between the end of the murmur and the beginning of the first sound (see Fig. 7). In these cases the jugular and liver pulses are invariably of the ventricular form, showing that the auricle, which erstwhile produced the presystolic murmur, is no longer acting.

Heart Failure in Mitral Stenosis

From the progressive nature of the lesions in the valve and in the heart muscle, it will be realised that the symptoms are not constant. The patient, as a rule, comes first under notice in early or middle adult life. The complaints, at this time, are shortness of breath, a sense of suffocation, and palpitation on exertion. In some instances the face is ruddy, with a hue a shade darker than is compatible with the ruddy countenance of robust health ; in others there is pallor. At this stage there is little or no increase in the size of the heart and no dropsy. A presystolic murmur can usually be detected. The patient's complaints may be the only evidence we have of the heart failure ; they point to an exhaustion of the reserve force. After a period of rest, this exhaustion may disappear, and the patient may go on for years with little further trouble. Some patients, however, break down again, when the symptoms complained of are usually those of the first attack. Frequently, however, a change is found in the character of the murmurs ; a diastolic murmur is now commonly noted, and there is sometimes a longer duration of the thrill. These signs imply an increased narrowing of the orifice. In those in whom no further narrowing takes place the murmur does not change. These are the people who go on for many years, and, if females, may bear children with no breakdown. We can thus infer that there is no progressive muscular or valvular sclerosis.

With the increased narrowing of the orifice, as indicated by the appearance of the diastolic mitral murmur, the heart becomes much embarrassed, the symptoms become much more distressing, and finally dilatation of the organ may set in. But even in the absence of progressive narrowing of the orifice dilatation may appear early. This is a sure sign that the rheumatic process has permanently injured the heart muscle. It is to be noted that the left ventricle also dilates in many cases, showing that there is some vital factor concerned in dilatation.

Heart Failure in Mitral Stenosis with Pregnancy

In mitral stenosis, as I have said, there does occur a degree of back-pressure which is the result of the narrowing of the mitral orifice. This latter event impedes the flow of blood so that there is a tendency to congestion of the lungs ; it throws, too, more work on the chambers behind, i.e. the left auricle, and right ventricle and auricle. When these begin to fail the circulation in the lung becomes embarrassed, and then in cases of pregnancy the growth of the uterus adds further to this embarrassment. I have dwelt already on the embarrassment which occurs in the pulmonary circulation as a result of the widening of the thorax and the impairment of its movements which take place in pregnancy. The early signs of this embarrassment are found in the crepitations at the bases (see Chapter VI).

But that is not the whole story. The damage due to rheumatic fever is rarely limited to the valves; it frequently extends also to the heart muscle. As a rule no certain evidence of this is available. We are therefore under the necessity of making an inference from the increased size of the heart, the great excitability of its action, and its marked inefficiency. Occasionally, however, we do get more direct evidence. This occurs when the damage invades the bundle connecting the auricles and ventricles, and produces a partial heart block. That point need not concern us here, for the heart block in these cases rarely, if ever, reaches such a degree as to cause embarrassment to the organ in its work. Its significance lies in the fact that it reveals an extension of the disease process from the valves to the wall of the heart.

The Estimation of the Significance of Mitral Stenosis in Pregnancy

Recognising, then, that the sources of danger lie in the narrowness of the mitral orifice, and in the embarrassment of the heart muscle, we are in a position to estimate the danger. Our estimate will take account of several considerations; it will depend in the main on *the rate at which the cicatrisation of the mitral valves is proceeding.*

I have already indicated the means of determining this in describing the changes which occur in the murmurs. These changes afford a basis of assessment, rough, it is true, but still of distinct value. I set them down in serial order thus:

(*a*) When, ten or fifteen years after the causative rheumatic attack, there is only a presystolic murmur, with no signs of œdema of the lungs, and the response to effort is good, then the outlook is favourable. Such an individual can be permitted to become pregnant with fair assurance of safety.

(*b*) When there is not only a presystolic murmur, but also a diastolic murmur. If the heart is normal in size and not too excitable, and if it is capable of a fair response to effort, then pregnancy may proceed. The patient, however, should lead a somewhat restricted life, avoiding especially such effort as brings on breathlessness or palpitation.

(*c*) When, even with a short presystolic murmur, and many years after the causation of mitral stenosis, there is marked inefficiency of the heart, shown by breathlessness on slight exertion, rapid pulse or easily excited palpitation, then there is danger in pregnancy.

(*d*) When the heart is large or irritable, and when effort readily induces palpitation and breathlessness, even if there be no diastolic murmur, then pregnancy should be forbidden. If, in spite of advice, it has been undertaken, the case should be carefully watched, particular attention being paid to œdema of the lungs. If crepitations become persistent after coughing or deep breathing, the advisability of inducing premature labour should be considered. If the percussion note of the lungs becomes impaired, interference is called for.

CHAPTER X

AORTIC DISEASE

Aortic Stenosis

The form of aortic disease which need concern us is aortic regurgitation. Aortic stenosis apart from regurgitation is so rare in the young that I, personally, have never met with it. I repeat here that a systolic murmur at the base—or as some might say " over the aortic area "—should never be looked upon as a sign indicating damage to the aorta; it is not infrequent in people with perfectly healthy hearts. Therefore a systolic murmur at the base of the heart, with a good response to effort and no increase in the size of the heart, is negligible.

Aortic Regurgitation

It is difficult to determine the relation of aortic regurgitation to heart failure.

I have watched many people with aortic regurgitation lead strenuous lives for twenty or thirty years with no limitation of their powers; others of my patients have suffered early from heart failure of a fatal kind. I have long endeavoured to distinguish between these types. I have found that in the former case the heart

has been little, if at all, enlarged and the blood-pressure only moderately raised. There has been no wide range between the systolic and diastolic pressures—in other words, there has been an absence of the " Corrigan " or " water-hammer " pulse. When, on the contrary, the heart is greatly enlarged and there is a marked " Corrigan " pulse (systolic pressure in the neighbourhood of 200, and diastolic 70 or 60), and when the response to effort is either greatly restricted or gradually becoming restricted, the outlook is grave.

I am inclined to the view that in the first type damage has been done only to the valves, and that to a small extent, while in the second the heart muscle has been invaded, or the valves have been greatly affected.

Heart Failure and Aortic Regurgitation

I must, however, confess that I have been baffled in my attempts to understand heart failure in many cases of aortic regurgitation. When this condition sets in, it does so in such diverse ways that clear comprehension is very difficult. Sometimes, for example, effort is restricted because of pain, and this pain may be of a severe kind and easily provoked. Yet the degree of pain seems to bear no relation to the gravity of the condition. Thus, I have found some patients suffer for long periods of years great pain on slight exertion, while in the case of others pain has heralded a fatal issue. In some,

again, heart failure is shown only by palpitation on exertion, these individuals suffering neither from pain nor breathlessness.

Some die suddenly with no previous sign of heart failure; others are as suddenly seized by an intense dyspnœa which ends fatally. Only a few have dropsy. I suspect that heart failure in these latter is due to the damage of the muscle more than to the regurgitation.

There is one peculiar feature about the heart failure of aortic regurgitation : when once it sets in, it proceeds apace. We all know what remarkable recoveries take place in people who break down from extreme heart failure with disease of the mitral valve, and even of the heart muscle; I have had no such experience in aortic regurgitation. My experience has been that once the heart gives way only very slight improvement ever takes place; moreover, in my own practice every case of aortic regurgitation who developed auricular fibrillation drifted to death.

Aortic Regurgitation and Pregnancy

My experience of aortic regurgitation complicated by pregnancy is limited to two cases. In these the heart was only moderately enlarged. One was prematurely confined (seventh month). Both made good recoveries, but were left very weak. In both palpitation was easily induced. Neither ever improved. They drifted and died within two years. I have, however, had under my care a number of cases with aortic regurgita-

tion who gave a history of rheumatic fever in their youth. They had borne children, and had not suffered in consequence. Presumably the aortic damage was done during the rheumatic-fever attacks. There was only slight enlargement of the heart.

Drawing from my experience in general, I would be disposed to allow pregnancy in a young woman with aortic regurgitation, if there was no " Corrigan " pulse, if the heart was not enlarged or only slightly so, and if the response to effort was good. On the other hand, if there was a forcible apex beat outside the nipple line, a marked " Corrigan " pulse, and a distinct limitation of the response to effort, I should decide that pregnancy ought to be forbidden.

CHAPTER XI

IRREGULAR ACTION OF THE HEART

The Significance of Irregular Heart Action

The importance of irregularity of the heart's action has not been sufficiently recognised. It is a matter seemingly so confused that few writers try to understand its real significance. Moreover medical knowledge had not until recently advanced far enough to enable physicians to differentiate its various forms.

This state of matters is unfortunate, because a clear understanding of irregularity throws light on many obscure features of cardiac action and gives information of the most valuable kind in diagnosis, prognosis, and treatment. Indeed the neglect of this subject renders a great part of the writings of physicians of little or no use. I have referred on p. 75 to Macdonald's description of cases of mitral regurgitation. In this description, as I pointed out, the incidental remark that the heart was irregular gives the clue to the real nature of the condition. An occasional reference such as this, if one is able to apply it, is often far more helpful than all the other details. Unfortunately, irregularity is usually considered so unimportant that it is not mentioned. Even

when it is mentioned, the reference is often so vague as to preclude an exact differentiation and diagnosis.

But there is more than this. It is unhappily true that one can rarely trust a statement that the heart was irregular. I have often seen perfectly regular hearts described as irregular. Tracings of these showed beyond possibility of dispute that irregularity was not present.

I go farther than that and say that even when a sphygmogram is given the statement that the heart is irregular cannot always be accepted. I have just been looking at some sphygmograms taken by a physician in a case of pregnancy. One of these is described as "irregular." Certainly a casual examination seems to justify this description, for the pulse waves are of varying height. But when the length of each beat of the pulse is measured accurately, this first impression is disproved. The beats are of exactly the same length and are absolutely regular. Thus the apparent irregularity is artificial. I know from my experience in taking records, especially when there is laboured breathing, that the movements of the patient's arm will cause a marked variation in the height and depth of the waves, which in this instance was mistaken for an irregularity.

When, over thirty years ago, I began the study of irregular heart action, I was told that the subject was so difficult and confused that it was a waste of time to attempt such an investigation, and that in any case little benefit would result

even if I happened to be successful. Even to-day, few physicians, and evidently no obstetricians, have made themselves familiar with this subject. As a consequence the subject is still shrouded in mystery ; and where we get mystery we get fear, and we find people with irregularities treated like people with murmurs. We have, too, the same kind of prognosis—"the sign may be of no significance, but, on the other hand, it may be serious." As there is no clear differentiation, there is no certainty regarding the forms which are innocent and those which are of serious import.

The Mechanism of Heart Irregularities

The vast majority of irregularities can be classified in a manner which renders them easy of recognition by the unaided senses. This, I think, is of great importance, for it is obvious that if knowledge of this subject is to be utilised in practice, differentiation must be accomplished without apparatus and in a reasonably short space of time.

The manner in which the irregularities arise will be made clear by means of the diagram (Fig. 8). This diagram represents certain structures concerned in the mechanism of the heart's movements. The heart's contraction starts at a small collection of peculiar fibres, the so-called sino-auricular node (*S.A.N.*) situated at the mouth of the superior vena cave (*S.V.C.*). The stimulus from the *S.A* node causes contraction of

the auricle (*A.*). From the auricle the stimulus passes to the auriculo-ventricular node (*A.V.N.*), the tissues of which resemble, in their structure, the fibres of the sino-auricular

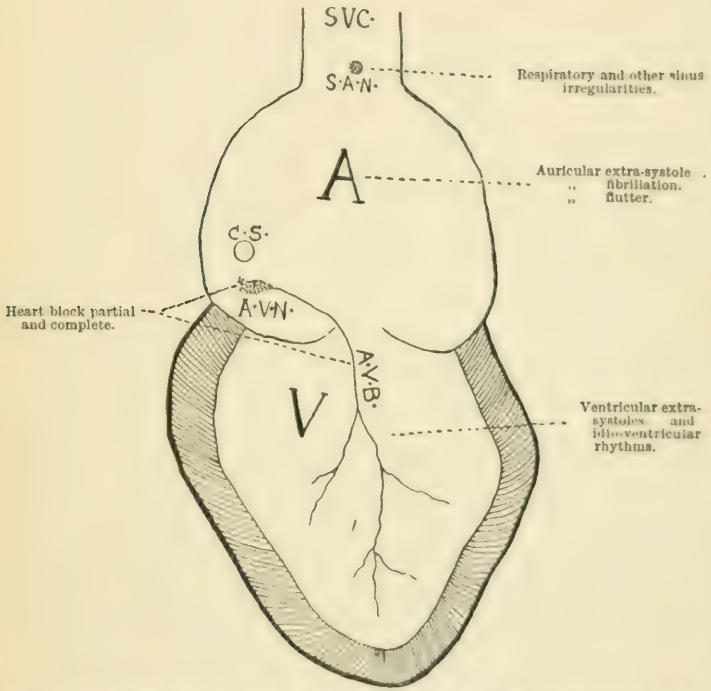

FIG. 8.—Diagram representing the parts concerned in the movements of the heart, and the places of origin of the different forms of irregular heart action. *S.V.C.*, Superior Vena Cava; *S.A.N.*, Sino-Auricular Node; *A*, Auricle; *C.S.*, Opening of Coronary Sinus; *A.V.N.*, Auriculo-Ventricular Node; *A.V.B.*, Auriculo-Ventricular Bundle; *V.*, Ventricle.

node. This *A.V.* node is situated in the wall of the right auricle near the opening of the coronary sinus (*C.S.*). The stimulus passes from the *A.V.* node along a bundle of peculiar cells, the auriculo-ventricular bundle

(*A.V.B.*) to the ventricle (*V.*), where it breaks up into innumerable branches distributed to different parts of the ventricle.

While, in normal circumstances, the stimulus of contraction passes in this manner, i.e. from the sino-auricular node to the ventricle, yet each of the different parts, the auricle, the *A.V.* node, the *A.V.* bundle, and the ventricle, is capable itself of initiating a stimulus which can produce a contraction. The reason that the stimulus arises first in the *S.A.* node is that this tissue is most sensitive. After a stimulus of contraction has been discharged, no other stimulus can be discharged until a certain time has elapsed. This time is required in order that the tissue may recover its excitability. The rate of recovery is most rapid in the *S.A.* node. Thus, other parts of the heart are again whipped to action before they are able to discharge a stimulus of their own manufacture.

This is why, for instance, in complete heart block, when the connection between the ventricle and the upper parts of the heart is severed (as in damage of the *A.V.* bundle), the ventricle beats at a much slower rate than the auricle. In this condition the auricle may be contracting at the rate of 70 or 80 beats per minute, while the ventricle pursues an independent contraction at the rate of 30.

In abnormal circumstances the parts below the *S.A.* node may become so excitable that the stimulus may arise in them and set up independent contractions, which anticipate in

point of time the stimulus for the *S.A.* node. This may occur occasionally in single beats, and so give rise to such irregularities as the extra-systole, which is due to premature contraction of the auricle or of the ventricle (Fig. 11). In the former case the auricular contraction precedes the stimulus from the *S.A.* node; in the latter case the ventricular contraction precedes the auricular or occurs at the same time. The premature contraction of auricle and ventricle may be continuous for a shorter or longer time, and so give rise to the various abnormal rhythms and the tachycardias (Figs. 20 and 21).

Differentiation of Heart Irregularities

The places where the different irregularities arise are shown in the diagram (Fig. 8).

In the course of an inquiry into irregular action of the heart, I found that there were several groups of cases, and that these could be clearly differentiated on two grounds:

(1) On the ground of the mechanism by which they were produced; and

(2) On the ground of their clinical significance.

While the first basis of differentiation has been the subject of much research, the second has received far too little attention. This is due, I think, to the fact that the method by which knowledge of the kind required can be obtained has not been understood.

It is perfectly evident that, while an understanding of the mechanism by which an irregu-

larity is produced is of profound interest and of great value, so far as the patient is concerned at any rate, knowledge of what is going to happen is of far more importance. To the doctor in charge of the patient such a knowledge is therefore essential to rational practice. This is particularly true in connection with the problem of pregnancy, where an abnormal sign like irregular action of the heart may be present and must be interpreted.

It will be found that in pregnancy we have to deal chiefly with three forms of irregularity: respiratory irregularity, extra-systoles, and auricular fibrillation.

Respiratory Irregularity

In this irregularity, which is probably the most common of all, the pulse is continuously varying in its rate. Simultaneous records of the radial artery and jugular vein show that the auricles as well as the ventricles participate (Fig. 9). While the irregularity can be clearly demonstrated by graphic records, it is necessary that the doctor should recognise it by his unaided senses. Advantage is taken of the fact that the alterations of the pulse are dependent on the respiration (Fig. 10). The pulse rate increases during inspiration, and slows during expiration. Frequently, however, this relationship cannot be recognised in a patient who is breathing normally; for a new stimulus has, in such cases, come into play before the effects of the previous one have

passed off. Thus a confused sort of rhythm is encountered. When, however, the patient is made to breathe slowly and deeply the relation-

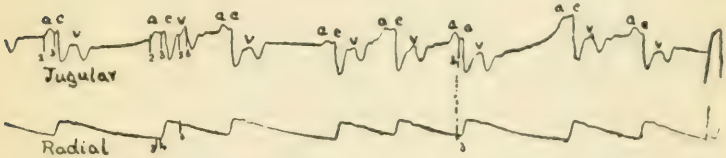

FIG. 9.—Simultaneous tracings of the jugular and radial pulses, showing an irregular pulse. The waves *c* in the jugular tracing are due to the carotid beat, and correspond in time to the pulse beats in the radial tracing. The wave, *a*, is due to the auricular systole, and it is seen that in this form of irregularity the auricles and ventricles equally participate—as shown by the waves *a* and *c*.

ship of the altering rhythm, with the different phases of respiration, can be easily determined (Fig. 10). I wish particularly to draw attention to this method of determining the type of

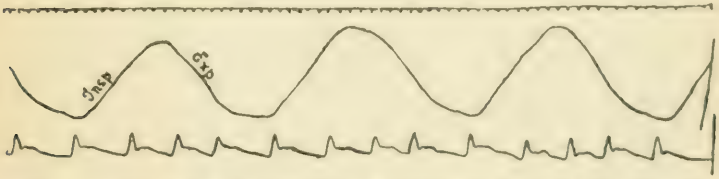

FIG. 10.—Tracings of the movements of respiration, taken at the same time as the radial pulse, showing that in the respiratory irregularity the rate of the heart increases during inspiration and becomes slower during expiration.

irregularity, as a doctor can apply it without any mechanical assistance. The character of the irregularity is best made out by listening to the heart at the same time as the patient breathes slowly and deeply.

The explanation of this irregularity is that a stimulus arises during expiration which affects the vagus and so produces the temporary slowing. On account of this fact the irregularity is sometimes spoken of as a "vagal irregularity," while on account of its association with the breathing it is called a "respiratory irregularity." Again, as it arises from a stimulation of the motor centre of the heart in the remains of the sinus venosus, it is called "sinus irregularity," to distinguish it from irregularities arising in other parts of the heart. There are, however, more than one form of sinus irregularity. I collected a large number of cases showing this irregularity, and found that the vast majority occurred in young people. The next most prevalent form of irregularity (the extra-systole) I found, on the contrary, to be most common in adults and elderly persons. So to make a distinction I called this "respiratory irregularity" the "youthful type," and the extra-systole the "adult type." I mention this differentiation as it has a distinct value in practice.

"Respiratory irregularity" is common in healthy young women, and in some women of maturer years, especially when there is present in the case a nervous element. It is a physiological phenomenon, indicative neither of disease nor of impairment. In no case should it be a reason for treatment or a bar to pregnancy.

The Extra-Systole

This irregularity is due to a premature contraction of the ventricle, occurring independently of the stimulus from the auricle. It occurs usually at the same time as the auricular contraction or a little before it. Sometimes the auricle also contracts prematurely; this premature

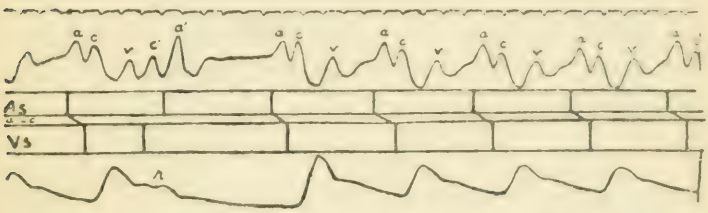

Fig. 11.—The upper tracing is from the jugular, taken at the same time as the radial (lower tracing). The interpolated diagram explains the events that happened in the heart during the period of irregular action. The down strokes in the space *As* represent the auricular systoles, and correspond with the *a* waves in the jugular tracing. The space *a–c* represents the passage of the stimulus along the *a–v* bundle, while the down strokes in the space *Vs* represent the ventricular systoles; and each corresponds with a pulse wave in the radial tracing. It will be seen that with one exception the auricular beat is followed by a ventricular beat. This one exception shows a wave *c′* in the jugular tracing, and a small wave *r* in the radial tracing, which occurs prematurely, i.e. before the auricular wave *a′*. These events represent what happens in the intermittent pulse, and show that the irregularity is due to a premature contraction (or extra-systole) of the ventricle.

auricular beat is followed by a small ventricular contraction. Premature beats can be recognised as small beats in the pulse occurring too early and followed by a pause. Sometimes the small beat is not felt, and there seems to be a long pause in the pulse. The irregularity can thus be recognised without graphic record by feeling the pulse and auscultating the heart at the same

time. When the pause in the pulse is felt the heart will give two short sharp sounds or one muffled sound, and this will be followed by a pause longer than usual (Fig. 12). A glance at the tracings and diagrams will explain the mechanism. If the pulsation in the jugular be observed during the pause of the pulse, a large wave may often be detected (Fig. 17). This is due to the fact that the auricular contraction is taking place at the same time as the premature ventricular contraction. The auricle cannot send its blood forward into the contracted ventricle, and so sends it back into the jugular vein.

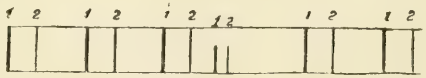

Fig. 12.—Diagram representing the sounds of the heart during an extra-systole.

Doctors should familiarise themselves with these clinical phenomena, as this form of irregularity and the "respiratory irregularity" constitute the great bulk of irregular heart actions. I repeat that both of them can, as a rule, be recognised by the unaided but trained senses.

My inquiries into the significance of this form of irregularity, which were pursued by watching large numbers of active people who showed it, revealed to me the fact that it is of no grave clinical significance. If heart failure did occur in people with extra-systoles, there were always other conditions present to account for it. So that, when this irregularity is the only abnormal

sign it can be ignored so far as the prognosis is concerned; if heart failure is present, then the prognosis should be based on the other signs and not on the extra-systole.

So far as pregnant women are concerned I found extra-systole present in 50 per cent. of healthy cases.[1] Usually they occurred at rare intervals and their detection was often accidental. Occasionally they were of frequent occurrence. In all cases the pregnancy was completed and the confinement passed with no trouble. I have in addition followed many of these women for twenty and thirty years, and the results justify this view.

[1] This was the percentage I found on examining 100 cases about thirty years ago. I have looked for my data, but I have lost the references and the notes of most of my cases. Among those of which I do possess records the percentage is not so high. The matter is not, however, of much consequence.

CHAPTER XII

AURICULAR FIBRILLATION AND PAROXYSMAL TACHYCARDIA

Auricular Fibrillation

The form of irregularity which is most commonly associated with heart failure in women at the child-bearing age is that due to fibrillation of the auricle. In this condition the muscle fibres do not contract in unison as in the normal beat, but each fibre twitches and contracts independently. The result is that the auricle is to all intents and purposes functionally inactive.

The Discovery of Auricular Fibrillation

It was this inactivity which led to the discovery of the condition. I had recognised that in mitral stenosis of a progressive type changes slowly took place in the murmurs (see Fig. 6, p. 82). I was therefore watching the course of the disease in a number of my patients whom I had attended many years earlier for rheumatic fever. In one case, a woman whom I had under observation for seventeen years, there had for many years been present signs of auricular activity; for example, a presystolic murmur, and the wave in

the jugular pulse, which I had come to associate with the beating of this chamber of the heart. There had also been present well-marked pulsa-

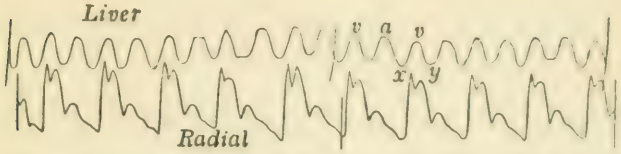

FIG. 13.—Simultaneous tracings of the liver and radial pulses in a patient with mitral and tricuspid stenosis, before the onset of auricular fibrillation. There are two waves *a* and *v* during the cardiac cycle (see Fig. 14).

tion of the liver, due, mainly, to a wave caused by the auricle.

The patient was suddenly seized with great breathlessness on exertion, and developed a rapid and irregular heart (Fig. 5, p. 58). When her heart had quietened down under digitalis I was

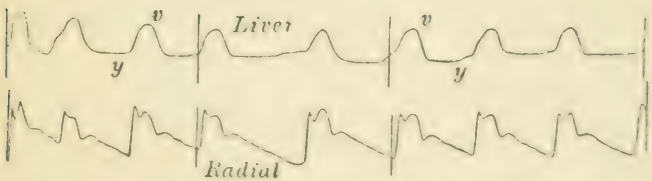

FIG. 14.—Simultaneous tracings of the liver and radial pulses after the onset of auricular fibrillation. There is but one wave now in the liver pulse during the cardiac cycle, the wave *a* having disappeared, and the presystolic murmur has also disappeared, and the rhythm is irregular. Compare with Fig. 13.

surprised to find that there was, now, no presystolic murmur, nor any waves due to the auricle in either jugular or liver pulse. In fact, at the time the heart had become irregular all signs of auricular activity had disappeared. I assumed that the auricle had become paralysed,

and in my early writings I described the condition as paralysis of the auricle (see Figs. 13 and 14, and also Figs. 6 and 7).

The Relation of Auricular Fibrillation to Extreme Heart Failure

The light thrown by this case led me to search for other cases with this irregular action, and to watch other patients who suffered from mitral stenosis. As a result I found the irregularity to be far from infrequent, and I was so fortunate as to detect its onset in a number of instances. This led me to a study of heart failure in its relation to this condition. I worked also to discover the best means of combating the trouble. There followed the discovery that probably 80–90 per cent. of all cases of heart failure with dropsy and enlarged liver are due to auricular fibrillation. Further I convinced myself and others that the astonishingly good effects of digitalis are practically confined to patients with this irregularity.

The Effect of Fibrillation of the Auricle on the Ventricle

The normal rhythm of the heart (due to the production of a stimulus in the sino-auricular node at the mouth of the superior vena cava) has already been described (page 95). When the auricle passes into fibrillation it ceases to contract as a whole, and the orderly discharge of the stimulus to the ventricle is interrupted. The

twitching muscle pours forth a rapid series of irregular stimuli, so that the auriculo-ventricular node is assailed by a great number of weak demands. Only certain of these—the actual number would seem to depend on the susceptibility of the auriculo-ventricular node and the bundle—pass through to the ventricle. The ventricular contraction is a normal one, but as a rule the rate of the ventricle becomes greatly increased, and the rhythm very irregular (see Fig. 5). There may, however, be exceptions.

Thus, in a case in whom for many years there had been a delay in the response of the ventricle to the auricle, due to a slight damage of the auriculo-ventricular bundle ("partial heart block") and in whom the rhythm was regular and the rate about 60, the onset of auricular fibrillation did not quicken the pulse. It did produce the characteristic irregularity. This patient was not conscious of the onset of the abnormal rhythm, and his response to effort was not impaired. In the vast majority of cases, on the other hand, the onset is not only accompanied by a great increase in rate, but also by a great limitation of the field of response to effort. Indeed, in eliciting the history of the condition one can often determine the moment of onset with great accuracy, for the patient will state that he was quite well until some effort—for example, hurrying up a hill—induced marked breathlessness. From that time onwards he has been breathless on slight exertion.

From the fact that the heart beats are not only

small and rapid, but also irregular, it can readily be understood how ineffective the circulation is bound to become (Fig. 5). Moreover, the rapid rate of the ventricle leads to exhaustion, so that heart failure speedily supervenes. This is in striking contrast to the absence of heart failure, when the rate is not increased as in the case just cited. When, in addition, there is a damaged ventricle, the rapid rate leads directly to a fatal issue.

Principles of Treatment in Auricular Fibrillation

Recognition of the fact, then, that the rapid rate leads to exhaustion of the heart muscle, and so to an inefficient circulation, gives us the data for applying a rational treatment. We must slow the heart. For even with auricular fibrillation the circulation is efficiently maintained when the rate of contraction is slow.

Study of the effects of digitalis and strophanthus in these cases showed that those drugs act by diminishing the rate in a most remarkable manner. I have repeatedly seen patients in dire extremity from heart failure (dropsy, orthopnœa, Cheyne-Stokes respiration), with a pulse rate of from 120 to 150, lose all their distressful symptoms under digitalis, and that speedily. The rate would fall to 60 or 70 beats. Relapses, too, which are apt to occur, could be warded off for years by the judicious administration of that amount of the drug which maintained the rate at from 60 to 80 beats when at rest.

The Recognition of Auricular Fibrillation by the Unaided Senses

The symptom by which the clinical observer can most readily recognise auricular fibrillation is the character of the pulse. The pulse rhythm is usually irregular, and this irregularity is of a very disorderly kind (Fig. 15, also Figs. 5, 7, and

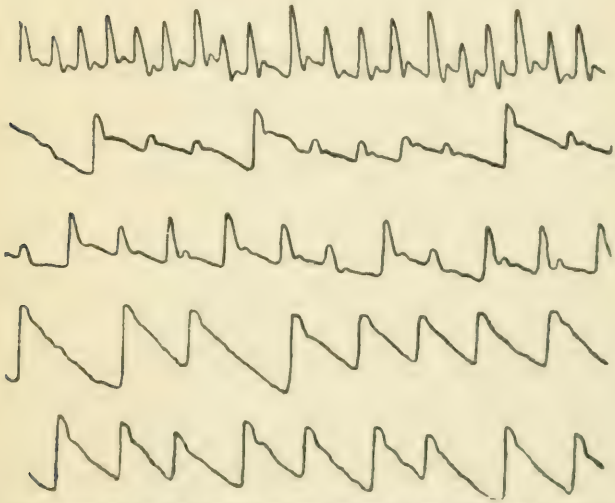

Fig. 15.—Tracings of the radial pulse, showing the disorderly irregularity characteristic of auricular fibrillation from five different patients.

14). In this respect it differs from most other arrhythmias. These, as we have seen, possess distinctive characters of their own. Thus, the irregularity in the heart of the young is marked by the fact that variations in rate coincide with phases of respiration; the intermittent pulse, due to extra-systoles, by the fact that the irregu-

larity breaks in on an otherwise regular rhythm. In auricular fibrillation the length of the pauses between the beats is, as a rule, continually changing, the pauses between two succeeding beats seldom being of the same duration; moreover, succeeding beats are rarely of the same strength. The character of these phenomena will be made clear by the radial tracings in Fig. 15. These tracings were taken from five patients.

Murmurs of Mitral Stenosis in Auricular Fibrillation

I have already stated that, with the onset of auricular fibrillation, all evidences of auricular systole disappear. Thus in a patient with mitral stenosis, whose condition has been revealed by the presence of a presystolic murmur alone, or by a presystolic murmur accompanied by a diastolic murmur, auricular fibrillation causes the presystolic murmur to disappear. The diastolic murmur remains, for the reasons already given. When the heart is beating rapidly the diastolic murmur may fill up the interval between the second and first sounds, and physicians are then apt to assume that it is presystolic in type. If, however, the heart be auscultated during one of the long pauses which frequently occur, or after the rate has decreased—for example, owing to the administration of digitalis—the murmur will be found to follow the second sound, and there will be noted an appreciable silence before the first sound occurring at the exact moment at which the presystolic murmur was wont to be

heard (see Figs. 6 and 7). This is a feature which even experienced clinicians seldom detect unless they specially seek for it.

The Bearing of Auricular Fibrillation on Pregnancy

My experience of pregnancy in women with auricular fibrillation is limited to half a dozen cases. This may seem a small number, yet if it be considered that these cases occurred at a time when I had a midwifery practice of forty or fifty confinements a year, it will be seen that altogether there must be a considerable number. All my cases gave a history of rheumatic fever; all had mitral stenosis. In each case the advance of pregnancy was accompanied by increasing signs of heart failure—limitation of effort and œdema of the lungs as already described (p. 49). In all but one premature labour set in between the sixth and seventh months. All lived through the confinement, but none ever recovered the same degree of health as was enjoyed before the pregnancy. I actually saw one patient in two pregnancies. She made a fair recovery. In the others heart failure increased gradually till they died within two years of the pregnancy.

Judging by my own experience, I believe that though pregnancy does not produce an immediately fatal heart failure, it so weakens the organ that it hastens the fatal issue. In saying this, I recognise that my experience is limited. At the time at which five of these cases were under my care I had not recognised the nature of auricular

fibrillation, nor had I learned how to give digitalis in this condition. In the one case which occurred after I had acquired this knowledge, I think I was able to help. The matter, however, requires far more investigation than I have been able to undertake. I leave it here, trusting that others, fortified by the knowledge now available, will carry out a more thorough inquiry.

In the meantime I would lay it down that auricular fibrillation is a bar to pregnancy. Should pregnancy have occurred, careful observation of the patient must be maintained. If the pulse rate exceeds 80 when at rest, digitalis should be given and continued until the rate falls to between 60 and 70 per minute. If œdema of the lungs, orthopnœa, or enlarged liver supervenes, the pregnancy should be terminated forthwith.

Paroxysmal Tachycardia

There are other abnormal rhythms which may have to be considered in relation to the pregnant state. The most common of these is a condition allied to auricular fibrillation and known as auricular flutter. In this condition the auricle does not contract in the usual manner, but beats ineffectively at a very rapid rate—usually about 300 times a minute. The ventricle does not, as a rule, respond to every beat of the auricle; it often responds only to every second beat, and sometimes to a varying number of beats, so that the pulse is slow and irregular.

The condition usually arises in paroxysms.

The patient is conscious of a sensation which suggests that her heart is "running away" at a great rate. This is accompanied by a sense of prostration. The paroxysm may last for a few minutes or a few hours. It subsides suddenly. A sudden onset and a sudden offset—the latter being followed by one or two forcible beats—are thus distinguishing features.

This form of paroxysmal tachycardia may occur with different types of heart trouble. Its significance in pregnancy should, therefore, be considered in relation to the presence or absence of disease. When there are no signs of disease, and the patient is well and strong between the attacks, the condition is not one of much importance. This is likely to be the state of affairs in young subjects. In the elderly, beyond the child-bearing age, the arrhythmia is often associated with disease of the myocardium, and may then be of serious significance. We may note, however, that paroxysmal tachycardia is not infrequently associated with mitral stenosis in quite young people. In such cases the physician should take into consideration what I have said in connection with that form of disease. The presence of auricular flutter naturally adds to the gravity of the condition.

I have had no experience of this abnormal rhythm as a complication of pregnancy. The opinion I have given is the result of the study of heart failure in connection with auricular flutter in a large number of cases, some of whom I have watched for many years.

Description of a Case of Abnormal Heart Action with Pregnancy

I have had one case of another and rarer form of abnormal rhythm. This woman went through

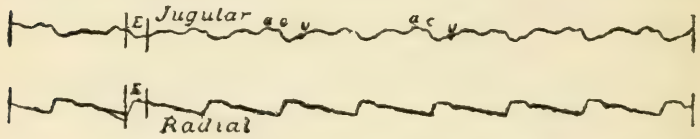

Fig. 16.—Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the auricular form.

her pregnancy unscathed. It is partly on the inferences which I drew from her that I formed the views I have just stated.

The woman (aged thirty-five) was pregnant

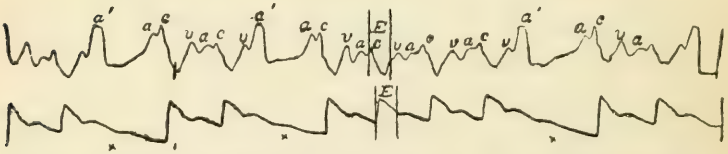

Fig. 17.—Simultaneous tracings of the jugular and radial pulses during irregular action of the heart. The auricle preserves its rhythm, there being a larger wave (*a'*) during the pause in the radial pulse. At this time there is a premature contraction of the ventricle, so that the auricular and ventricular contractions occur at the same time, and the contents of the auricle, not being able to enter the ventricle, are thrust back into the jugular, causing the larger wave *a'*.

with her first child. For several years previously she had suffered at times from breathlessness and irregular action of the heart. During the few weeks in which she was under my care (at the eighth month of pregnancy) the pulse was

usually irregular—the degree of irregularity varying very much. When the heart was irregular the venous pulse was always large, while when the heart was regular it was barely perceptible, so much so that it was with difficulty that I could

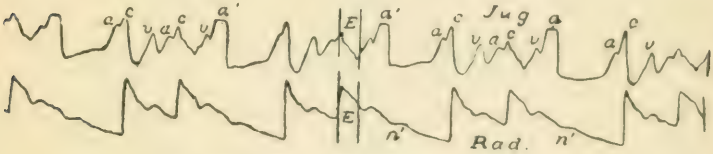

Fig. 18.—Shows the same thing as Fig. 17.

get a tracing of it (Fig. 16). Figs. 17 and 18 show the different characters of the irregularity. In Fig. 17 there are long pauses in the radial pulse (× × ×). During these the auricular wave *a′* becomes large, an evidence that at this time the auricle and ventricle contracted together. The

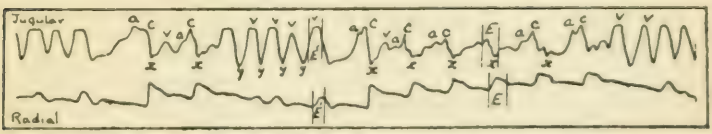

Fig. 19.—Shows a number of normal beats, with the usual waves, *a*, *c*, *v*, in the jugular, and a series of abnormal beats, small and rapid, with the waves *v* in the jugular. These abnormal beats are due to a series of independent ventricular contractions.

ventricle, however, was in systole (an extra-systole), and so the contents of the auricle could not be sent into the ventricle. They were therefore thrown back into the jugular vein. Hence the large wave *a′*. The extra-systole does not appear in the radial pulse. It is a premature contraction of the ventricle (a ventricular extra-

systole). This was so small that it did not send any wave into the radial artery.

Occasionally this heart would break into a

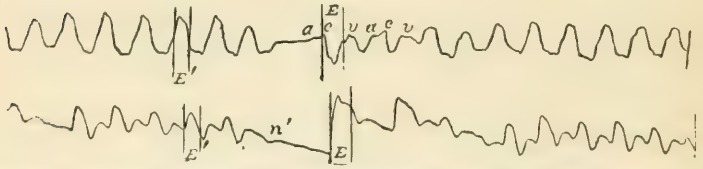

Fig. 20.—Shows two normal beats in the centre, all the rest are independent ventricular beats.

series of rapid beats as shown in Figs. 19, 20, and 21. Thus there was produced a form of tachycardia whose origin is of the same nature as that of the premature ventricular beats in Figs. 17

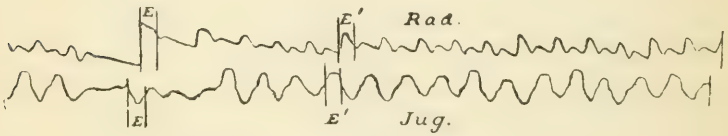

Fig. 21.—Shows only one normal beat (*E*).

and 18. I did not attend this patient in confinement, but I heard that she passed through it without trouble; some years after I heard that she was quite well.

CHAPTER XIII

THE NEUROTIC HEART

By this term I refer to a very distinct type of heart affection in which the symptoms are due mainly to disturbances of sensation. The patients complain of pain in the left chest, usually under the breast. The pain is of a dull aching character, accompanied sometimes by "stabs." The aching varies in severity, slowly increasing and decreasing. It may last for hours; or, again, the patient may be conscious of it for days. It is not usually immediately aggravated by effort; but it may appear some hours afterwards, or, if present before, may become worse when effort is undertaken. Only occasionally is it provoked to a severe degree by effort. Attacks of great severity resembling attacks of angina pectoris sometimes arise. In these instances doctors may alarm their patients and their patients' friends by telling them that the condition is in fact angina pectoris.

It should be recognised further that these attacks of pain may come on with great severity in the absence of any apparent provoking cause.

Site of Pain and Associated Phenomena

The distribution of the pain, while mainly confined to the left chest, may extend also into the arms and up the side of the neck. The attacks are often preceded or followed by a sense of chilliness, or cold. They are frequently accompanied by shivering resembling a rigor. There is usually present hyperalgesia of the skin and deeper tissues of the chest wall. This can be elicited by lightly pinching or pressing the left breast. In a less degree the right breast also is tender to pressure. The aching of the breast and its tenderness may lead the patient to think that there is some disease in this region.

These patients are liable to attacks of palpitation; and the heart's rate is sometimes unduly increased in response to effort or excitement.

When asked what sensations stop them while walking on the level, they usually reply: "A feeling of exhaustion, with a desire to sit or lie down."

The Relation of the Neurotic Heart to Physical Signs

The phenomena described above occur frequently in women; and the hearts of these women show, as a rule, no abnormal physical sign. In some, however, a systolic murmur may be detected, or an irregularity of the respiratory type or extra-systoles. The size of the heart may be slightly increased.

Those abnormal signs do not in any sense add to the gravity of the state; they are merely incidental. The condition, in short, is not due to disease of the heart—though it may be associated with disease, as, for example, mitral stenosis. It is due to some agent which increases the excitability of the whole nervous mechanism of the heart. Pregnancy can safely be undertaken by these people. It often does them a great deal of good.

Conditions predisposing to the Neurotic Heart

There are three conditions which induce this form of cardiac weakness: (1) toxic states; (2) overwork; (3) sexual disturbances.

Toxic states.—The source of poisoning is most frequently an intestinal trouble. In every case of this sort a careful inquiry should be made into the condition of the digestive organs. The abdomen should be examined for signs of cutaneous hyperæsthesia and muscular rigidity. The presence of these latter is a sure indication of some old-standing trouble in this region. Appendicitis, gastric ulcer, adhesions around the cæcum or gall bladder, and intestinal stasis, are among the most common conditions found. Sometimes the source of infection may be an abscess in some other part of the body, as, for example, in the antrum or at the roots of the teeth.

Overwork.—Women who have too long hours

of labour, with little sleep, are apt to develop nervous hearts. The long nursing with disturbed nights of an ailing parent, husband, or child by a woman who has to do her housework or earn her living is an illustration in point. Any form of fatiguing work with mental worry is apt to induce the condition.

Sexual disturbances.—When no apparent cause which would account for the condition can be found, inquiry should be made into the sexual functions. For it is a fact that this type of heart is apt to be engendered in otherwise healthy young women who are married to impotent men or men who are only capable at long intervals.

One of the most extreme cases of this type which I have seen was a woman of twenty-six, who had been married for two years to an impotent old man. Another case was a young woman who had had no intercourse with her husband, with whom she slept, because her doctor, a maiden lady of mature years, had warned her that this would be dangerous on account of her weak heart. Complete recovery followed the assumption of normal relations with her husband, and pregnancy followed.

The neurotic heart is also frequent among women, whether married or not, who indulge in masturbation. The worst case I have seen was a pretty young Jewess, whom I examined some weeks after the birth of her first baby. Not only were there present the signs of neurotic heart already described, but the heart rate was persistently increased to 130–150 beats per minute.

She confessed to indulging in the habit six or eight times a day.

While dealing with this subject I may refer to another matter which bears a relation to it—the danger, or supposed danger, of coitus to women with damaged hearts. The domestic happiness of married couples is often marred by the injudicious advice of doctors who have, on account of fears as to the hearts of patients, forbidden intercourse. I have just cited an instance of the unwisdom of this advice; I have found it given so frequently that I wish to state that I know of no heart condition which should act as a deterrent, when the woman feels competent. It is a matter for the woman, and not the doctor, to decide. If she finds that it comforts her, as it often does, and if she experiences afterwards nothing but a passing exhaustion, there is no ground for objection, however affected her heart may be.

CHAPTER XIV

CONGENITAL DEFECTS OF THE HEART AND PREGNANCY

General Principles

I have not had much experience of the complications of pregnancy by congenital defects of the heart. The opinions which I express, therefore, are based only on a knowledge of general principles. I would suggest that in doubtful cases doctors should be guided by a recognition of the state of the heart's efficiency. When the organ is enlarged, or when there is cyanosis or clubbing of the fingers, the response to effort will be so limited that it will readily be seen that pregnancy is a danger and should be avoided. When, on the other hand, the heart is normal in size, or only slightly enlarged, the response to effort good and no cyanosis present, then, notwithstanding any physical sign—such as a murmur—which may be found, marriage and pregnancy can be allowed.

Types of Congenital Murmurs

There are two congenital murmurs which stand out prominently. The first, and most common, is a loud, rough, systolic murmur,

heard all over the heart, the area of maximal intensity of which it is difficult to determine. I do not know for certain where this murmur arises, though I lean to the view that it has its source in a small communication between the ventricles. I have met it so frequently in the young, and so rarely in the adult, that I suspect that it disappears with growth. I have, however, never been able to follow my cases long enough to see.

Patent Ductus Arteriosus

The other murmur is due to a patent ductus arteriosus. It is a remarkable murmur, and when once recognised can always be identified easily. The tube connecting the aorta and pulmonary artery remains in these cases patent after birth The pressure in the aorta being higher than that in the pulmonary artery, the blood passes through during the whole of the cardiac cycle and produces a continuous murmur, which is loudest at the beginning of systole, and which diminishes in loudness during diastole. It is always accompanied by a thrill over the second and third ribs on the left side. At this place the murmur is loudest.

I have only seen one case of patent ductus arteriosus in a pregnant woman. In this instance I was called into consultation to decide whether the pregnancy should be allowed to proceed and whether the patient should take chloroform. I had no hesitation in recommending that the pregnancy should be allowed to go to full term,

and that chloroform should be freely given. The woman came through her confinement, and through a subsequent one, with no unfavourable incident.

I have seen a considerable number of cases of patent ductus arteriosus in the young, but only three or four in persons over twenty years of age. I have not been able to follow up my cases; but it occurs to me that this condition also may disappear in adult life.

CHAPTER XV

THE MANAGEMENT OF CASES OF PREGNANCY WITH HEART DISEASE

With an Efficient Heart

The first duty of the physician is to decide whether or not there is any reason to expect heart failure if pregnancy occurs. The presence of abnormal signs gives no indication of the functional efficiency of the heart. That must always be estimated on the lines laid down above. If the functional efficiency is not impaired to an extent greater than that occurring in a normal pregnancy, no steps need be taken and no anxiety felt.

When, however, a woman with undoubted heart disease, such as mitral stenosis, does become pregnant, the pregnancy should be allowed to continue only so long as no marked signs of heart failure are present. Such exercise as can be undertaken in perfect comfort may be permitted. But any distress, particularly breathlessness, is an indication that effort must cease.

With an Inefficient Heart

The patient should be examined weekly for signs of heart failure, particularly for signs of

œdema of the lungs as described on page 49. If crepitations at the bases of the lungs appear, persist, and tend to increase, they may be taken as evidences of the onset of heart failure. There will also, in such cases, be present a great limitation of the field of response to effort—breathlessness being induced on slight exertion. The patient should be confined to bed or couch, and her activities reduced to a minimum. She should be encouraged to sit up or lie propped up in bed, since lying down, by restraining the movements of the ribs, tends to hamper the circulation in the bases of the lungs. At times, during the day, she should be made to breathe deeply, in order to assist the right heart in expediting the flow through the lungs.

The Management of the Labour

If heart failure is kept in check the pregnancy can be allowed to go to full time. It often happens that labour sets in, in these cases, about the seventh month; so that, as a rule, the birth is accomplished with little stress on the heart. It was my custom to give chloroform at an early stage of labour—light anæsthesia at first. My object was to restrain the "bearing down," which taxes the heart heavily. When the labour had advanced so far as to justify the use of forceps I pushed the chloroform to complete anæsthesia and delivered. In this way the straining of the last stage was avoided.

I gave chloroform freely to my midwifery

cases, and never had any trouble. It is possible that "twilight sleep" may be better than chloroform, but I have no experience of it.

Indications for Shortening the Pregnancy

When the heart failure is so extreme as to threaten life, intervention is necessary. Premature labour or miscarriage should then be induced.

Those patients in whom I induced miscarriage in the third or fifth month suffered from mitral stenosis. I had attended them in previous confinements which they had come through with difficulty—the heart failure being so extreme that it was manifest that another pregnancy would probably prove fatal. In nearly all my cases of mitral stenosis who carried to the later months premature labour set in spontaneously.

Medicinal Treatment

Medicinal treatment by drugs of the digitalis group is not satisfactory unless auricular fibrillation is present. I have rarely found any benefit from these drugs in heart failure in mitral stenosis, so long as the rhythm remained normal. If the rate of the heart is much increased, however, digitalis may be given a trial, for sometimes it does slow the heart when the rhythm is normal. It is in auricular fibrillation that the good effects of the drug are obtained. Here heart failure is nearly always associated with a rapid rate, many of the beats being ineffective. Digitalis slows

the heart, and the individual beats become strong and effective. The circulation is thus at once greatly improved. When the heart responds to digitalis, and the rate falls to 70 or 60, the drug should be suspended, and only resumed when the rate begins to increase. The quantity sufficient to keep the rate about 70 should be determined by giving and withholding the drug during a period of a few weeks. Thereafter the patient should continue for the rest of her life to take that quantity which keeps her heart beating at a moderate rate.

I have found the tincture of digitalis so satisfactory that I rarely recommend any other form. The dose should be fifteen drops four times a day, and the effect should be looked for on the fourth to the seventh day. The symptoms of overdose are nausea or vomiting, or diarrhœa, or a tight feeling of distress across the chest or a great fall in the rate. When any of these symptoms appear the drug should be stopped at once. It should be cautiously resumed when they disappear. In this way digitalis can be given without fear of untoward result.

General Management

It is needless to dwell upon the general management of such cases. Patients must, of course, avoid conditions which throw a strain upon the heart—as, for example, mental worry, straining at stool, and digestive troubles. In regard to digestion, flatulence is often a distressing

complication. It can best be avoided by taking meals in a dry state so as to compel thorough mastication. Meals should also be small in quantity and frequent.

Sleep must be obtained during the pregnancy. If it does not come naturally, then the milder hypnotics should be given, beginning with the bromides. If these fail, some of the tar products should be tried. If, in spite of these, sleeplessness persists, chloral or opium, or one of its derivatives, may be cautiously administered.

CHAPTER XVI

SUMMARY

As the danger attending pregnancy in women with heart disease is the occurrence of heart failure, the physician must keep clearly before him the symptoms by which this can be recognised.

The absence of a clear conception of the nature of these symptoms has too often led to a misunderstanding of their significance. No sign manifested by the heart itself gives information as to the functional efficiency of the organ, and consequently the signs of heart failure must be looked for in other directions and more especially in those structures whose blood-supply is likely to be reduced by the weakening of the circulation.

Extreme heart failure is shown by such signs as dropsy, enlargement of the liver, œdema of the bases of the lungs, or cyanosis.

Early heart failure may be revealed by no sign when the body is at rest, and may only be discovered by distress evoked when some effort is made which the patient was wont formerly to perform in comfort.

The signs of distress, so far as women in the child-bearing period are concerned, are breath-

lessness and palpitation. Pain on effort may be present in certain cases of mitral stenosis and aortic disease; but, as a rule, the pain of grave heart failure is a sign which occurs much later in life.

From this it follows that no single sign shown by the heart itself, however abnormal it may seem, should be a bar to pregnancy.

Systolic murmurs, no matter in which area they are loudest, should never be a cause of anxiety in pregnancy. If they occur in hearts which show no other abnormal sign, and if the patient's response to effort is good, they should be ignored. If they are associated with other signs of heart disease, the prognosis should be based on these other signs and not on the systolic murmurs.

The same rule applies to the irregular actions of the heart due to respiratory arrhythmia and extra-systoles.

In women with easily excitable hearts, who suffer at times from pain of varying degrees of severity (the neurotic heart or the toxic heart), when the organ is normal in size, or only slightly enlarged, the heart trouble constitutes no bar to pregnancy. This applies whether systolic murmurs are present or not.

The form of heart disease which gives most occasion for anxiety in pregnant women is mitral stenosis. This usually has followed an attack of rheumatic fever. In such cases great care must be taken to differentiate between dangerous and not dangerous forms of the malady. The

latter include instances in which the cicatrising process, which produces the stenosis, is stationary or is only progressing slowly. Such slow progress is shown by the character of the murmur. If there is present ten or fifteen years after the causative attack of rheumatic fever only a presystolic murmur, and if the heart's size is normal, its rate regular, and the response of the patient to effort good, then pregnancy may be undertaken with fair prospect of safety.

If, on the other hand, there is present, within a few years of the causative attack of rheumatic fever, a diastolic murmur as well as a presystolic, there will be danger. This will be particularly the case when there is evidence also of the heart muscle being damaged, i.e. enlargement of the organ and much distress on effort. When in addition to the mitral stenosis there is fibrillation of the auricle, pregnancy should be forbidden. If pregnancy has taken place, then the case should be watched; and if grave signs of heart failure occur, the pregnancy should be terminated.

In cases of aortic regurgitation, if the heart is normal in size and the response to effort is good, pregnancy may be undertaken. If, on the other hand, the ventricle is much hypertrophied, and there is a marked "Corrigan" pulse, the probability is that the heart will be so permanently impaired that it will cripple the patient severely if she gets over her confinement.

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